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STUDIES FROM THE

Department of

NEUROLOGY

1922/24

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477 FIRST AVENUE
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STUDIES FROM THE Department of NEUROLOGY

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DEPARTMENT OF NEUROLOGY

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THE MODERN AND TECHNICAL STUDY OF HEREDITY*

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Heredity is a name given to a biological process by which the characters of body and mind are transmitted from parent to offspring. The study of heredity includes a study of the biological process itself, which is an analytical and synthetic attack, and a study of the clinical phenomena of heredity, which is a descriptive attack.

Very properly, yet only recently, heredity has been taken up by the biologist and become a laboratory study. In the biological method of studying heredity, there have developed new terms and a modern discussion on heredity involves almost a special vocabulary. It is my purpose to indicate the lines of work that are being followed just now in these biological studies of heredity and to give such descriptions of laboratory work and terms as may help to interpret our clinical observations and make them contribute effectively to the science of heredity as a whole.

Heredity is one phase of the more general subject of genetics, a science which deals with the coming into being of organisms and which includes not only parental influence, but all environmental influences which in some way affect the bringing into being of a new organism or race (1).

Many students of heredity enlarge their range and call themselves geneticists.

And it would be wise for us who are studying

*Read at a meeting of the Association for Research in Nervous and Mental Diseases, December 26, 1923.

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the inheritance of nervous and mental diseases to take the attitude of the geneticist and consider the factor of environment also in so far as it relates to our subject.

In accordance with the definitive character of my remarks, I would like to state what I think should be clearly in mind, when we speak of inherited disease:

Inherited morbidity shows itself in three ways, viz., as inherited organic diseases, as inherited functional disorders and as inherited constitutional predispositions. Inherited morbidity in all these forms must be an inborn heredity. Diseases started during prenatal life, by trauma, infection or malnutrition, are not technically hereditary through these causes. In this sense, there is really no hereditary syphilis or hereditary tuberculosis. But there may be inherited predisposition to these and many other morbid conditions. Inherited abnormalities and defects are often classed as inherited disease and an exact distinction can not always be made. Thus many of the familial diseases might be called abnormalities or mental defects.

Abnormalities of development are often produced by some accident during fetal life, and are not to be classed as inherited disease unless they are passed on later in the form of familial diseases. The fact that particular abnormalities are not necessarily inherited is to be considered in studying hereditary monstrosities, and defective neuropsychic characteristics. Feeblemindedness, for example, is probably often due to a prenatal injury, not to an inheritance and is not necessarily transmitted.

BIOLOGY AND GENETICS

In studying the inheritance of disease, we must pay due weight to the work of the biometricians and the school of Galton. At one time there was great antagonism between the latter group and the Mendelians. It has been stated that, according to Pear-

son, no inheritance is Mendelian; while according to Davenport all inheritance is Mendelian. Of late this situation has somewhat changed. The grounds covered have become somewhat different. The biometricians deal with statistics and mathematics in relation more especially to the general problems of population and social life. The Mendelians are dealing with the living cell and its mechanisms in relation to heredity. The biologists in studying heredity seem now for the most part to be working along Mendelian lines. Their work involves a great deal that is highly technical and it has adopted a stupendous vocabulary. It is, however, extremely interesting and is a vital phase of the science of inheritance. So I would like to refer to some of its data and methods.

In the substance of the cytoplasm of a plant cell is a nucleus which contains a chromatic network that breaks up into granules and pairs of bodies called chromosomes. Every cell of the body has a constant number of paired chromosomes, and they are one half from the male and one half from the female. They are supposed to carry "factors" that determine the characteristics of heredity, e.g., tallness, shortness, albinism, pigmentation, etc. This special factor is called a "gene" or "id." It is of the nature of an enzyme, secreted by the nucleus. A gene has no anatomy and no chemistry; it is spoken of as a "mood"; but it is a structural peculiarity in the chromosome and it occupies a particular part of the chromosome. A gene for color is one which causes pigmentation, while in the absence of this color gene there is albinism. Changes in the genes are the cause of variability in inheritance or "mutation" (2). There is a number of these genes in a chromosome; they vary in character and this explains blending of inheritance. Thus where a black rabbit is mated with a white one, a gray colored rabbit results, there being at least five different kinds of genes in the chromosomes.

It is believed that chromosomes never fuse absolutely, and on this fact it is inferred that individual characters are not actually blended, but that the hereditary characters making up a personality, or a defect or a disease, are the results of the segregation of individual unit characters, anatomical or functional.

MENDELISM

These biological data bring us to the subject of Mendelism, which seems to be widely accepted now as the basis of the biological mechanics of heredity. The essential features of Mendelism as it affects our problems are, that an organism is made up of unit characters and that a real blending does not take place in the reproduction of offspring. Hereditary characters, to put it more explicitly, are independent units (usually) which segregate upon the fertilization of the female by the male, regardless of temporary dominance. (2). This general statement can be elaborated into two specific laws:

First, the law of dominance: When two organisms of different unit characters are crossed, hybrids result and these hybrids are all apparently alike and have the character that is dominant in one of the ascendants, i. e., they are all tall or short, etc.

Second, the law of segregation or disjunction: If these hybrids are bred together, their offspring, forming a third generation, will consist of fifty per cent. which are like their hybrid parents, twenty-five per cent. like the male procreator and of pure stock, twenty-five per cent. like the female and also of pure stock. If S represents the original male and s the female, the formula for the grandchildren is S+2Ss+s.

The main points viz., that of the unit character of inheritance and of the influence of the dominating and recessive qualities, may be shown in the following illustrations: Assume that: M is a dominant character (assume it to be "hereditary chorea").

Assume that N is a recessive character (i.e., "normal neuromuscular mechanism"). Now if one breeds M N with N N there results fifty per cent. M N plus fifty per cent. N N, which means one half are sick, one half well, or if N is dominant all would be well.

Suppose further that M is recessive and N dominant, then breeding two hybrids M N with M N gives twenty-five per cent. M M plus fifty per cent. M N plus twenty-five per cent. N N equal one half sick, three quarters well. (See *Apert L'Heredite morbid*, 1919.)

The data concerning inheritance have been worked out especially in plants and lower animals. They apply in a measure to certain special human characters that may be considered unit characters, such as pigmentation, polydactylism, and perhaps other anatomical characteristics. Tallness and shortness are unit characters in the pea, but in man tallness involves the internal secretions and conditions of nutrition and metabolism that make their quality quite complex. The familial diseases, being really defects more than diseases, are supposed to show Mendelian traits. In fact Mendelism can be applied perhaps more definitely to human defects than to disease.

But on the whole, neurologists and psychiatrists must at present expect to get most help in studying inheritance of disease from the biometrician, from clinical observation and a study of the environment. The application of Mendelian laws to the inheritance of human disease means a long wait.

The following are some of the technical terms in connection with Mendelian heredity:

An allelomorph is a pair of characters of which one is dominant and one recessive. When such a pair is bred, the result is 1+2+1, 1 being pure dominant, 2 hybrid, 1 pure recessive.

A gamete is a mature male or female cell ready for maturation. A gamete is a marrying cell.

A zygote or yoked cell is a fertilized cell formed by the union of two gametes; it is a married cell.

The gene is the factor in the chromosome invisible and undemonstrable anatomically or chemically which gives form and substance to a definite visible unit character in an organism.

A homozygos character is one which is present in the offspring in double quantity because it was present in both parents.

A homozygote is an organism which is homozygous (double dose) with respect to any character.

A heterogote is an organism which derives the determiner of its character from only one parent.

NOSOLOGY OF INHERITED DISEASES

I would now like to call attention to the classes of nervous and mental disease to which laws of inheritance are to be applied; they are: Familial diseases, or diseases of similar heredity. These are diseases which have been evolved into a family and become part of its chromosome pattern. Familial diseases are those which tend to create out of apparently normal individuals a type that is abnormal and one that is degenerate in character.

The familial diseases are characterized as follows:

a. There habitually develop several subjects of the diseases in the same generation.

b. The cases develop in the same way and with the same characteristics.

c. They develop out of a constitutionally neuropathic constitution and independent of any external pathogeny.

d. They are rarely cases of direct heredity, i. e., passing from father to son, etc.

The principal familial diseases are: Heredity ataxia of Friedreich; congenital familial bleheroptosis (Moebius); familial spasmodic paraplegia; amaurotic idiocy (Tay-Sachs); familial or hereditary optic neuritis; the progressive myopathies; familial periodic paralysis

(Westphal); familial nocturnal enuresis; essential or familial polyuria; essential hereditary tremor; chorea of Huntington.

Apert makes the following groups of hereditary nervous diseases:

Familial diseases with a continuous heredity—Here the disease is transmitted from parent to child and never skips a generation, e. g. Huntington's chorea.

Fraternal familial disease—The patients are the children of healthy parents, but one finds the disease in a collateral ascendant like an uncle.

Diseases of matriarchal heredity—Here descendants of affected males always escape, and only those descendants of the common stock on the mother's side are affected. In some cases the disease is perpetuated in continuity from mother to child, the daughters always carrying the malady. This is seen in familial periodical paralysis and Daltonism. The disease also can be perpetuated by the female who does not have the disease, as in familial papillary atrophy or hereditary optic neuritis. In these cases the disease has the character of a dominant.

DISEASES OF DISSIMILAR HEREDITY

In diseases of dissimilar heredity, there is a transmission of a morbid aptitude, i. e., a constitutional predisposition to a nervous disease, but the disease transmitted may be the same or may be different in the offspring from that of the parent. Illustrations of this are families in which there may appear one or other of such maladies, as epilepsy, migraine, the psychoneuroses, Basedow's disease, and family tremor, the constitutional psychoses, such as those of the manic depressive and dementia precox types, morbid alcoholic craving and similar constitutional states of dependence on defective inhibition. The mechanism of the transmission of these morbid aptitudes, is one of the objects of biological research.

There is a large class of diseases in which poisons

or infections during prenatal life induce enfeebled or neuropathic constitutions, which make the descendant liable to acquire the parental disease. Such diseases are tuberculosis and syphilis. These are not cases of the heredity of nervous diseases and their study comes more properly under the head of congenital disease.

It seems, however, possible that defects or anomalies in the nervous mechanism produced prenatally by certain accidents, may be inherited, indeed it is only in this way that we can explain the origin of the familial diseases.

Characters acquired prenatally, may be transmitted but, despite Prof. Kammerer, is seems to be still pretty well agreed that in the higher animals, characters acquired postnatally are not inherited.

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TESTICULAR TERATOMA WITH SECONDARY DEPOSITS IN SPINAL COLUMN AND MENINGES*

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AND

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NEW YORK

This case is presented because of certain uncommon features in its microscopic examination, some of which explained a confusing clinical picture:

T. C., a man, aged 38 years, was admitted to the Neurological Department, Bellevue Hospital on February 14, and died there thirteen days later. Syphilitic infection occurred at the age of 20. The first symptom of his illness was in October of last year when he experienced a series of severe girdling pains in the distribution of the first lumbar roots. These pains were felt at fairly widely separated intervals up to two weeks before admission to the hospital. He then noticed a weakness of the left leg which in fifty-six hours was useless; paralysis of bladder and rectum followed quickly, and in three more days there was total flaccid paralysis of both lower extremities and of the lower trunk muscles, together with a loss of all sensibility from the level of the fifth dorsal segment downward. There were poorly defined sensory losses above this level.

The abdominal reflexes and those in the legs were abolished. Sacral bedsores speedily developed. The spinal fluid was yellow, most rich in globulin, and two good observers reported 975 leukocytes in each cubic millimeter.

An enlarged left testis was thought to have been the product of an old orchitis. The blood was strongly positive to the Wassermann test. An extensive laminectomy in the lower cervical and upper dorsal region revealed a dry, triangular cord, but no sign of the expected abscess. The patient died on the following day.

Dr. Douglas Symmers performed the necropsy and we are indebted to his courtesy for the necropsy report of which the following is a condensation: The left testis was hard and enlarged; on section a whitish nodule 3 cm. in diameter was found in it. The liver contained several subcapsular whitish areas, and some greenish gelatinous nodules which appeared to be entirely encapsulated. Lying on the left anterolateral aspect of the vertebral column and beneath the pleura was a mass of tissue extending from the second to the seventh dorsal vertebra. It measured 12 cm. long, 8 cm. wide and 7 cm. in thickness. It was deep red, mottled with whitish patches, and soft. The diaphragm was similarly mottled and infiltrated.

The spinal cord in the region of the tenth dorsal vertebra was adherent to the anterior surface of the canal. The bone was here eroded. The spinal tissue below this plane (Fig. 1) was entirely necrotic.

The diagnosis of spinal cord abscess was reached through a consideration of the clinical course and the high count of what were taken

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* Read before the Forty-Ninth Annual Meeting of the American Neurological Association, Boston, May 31, 1923.

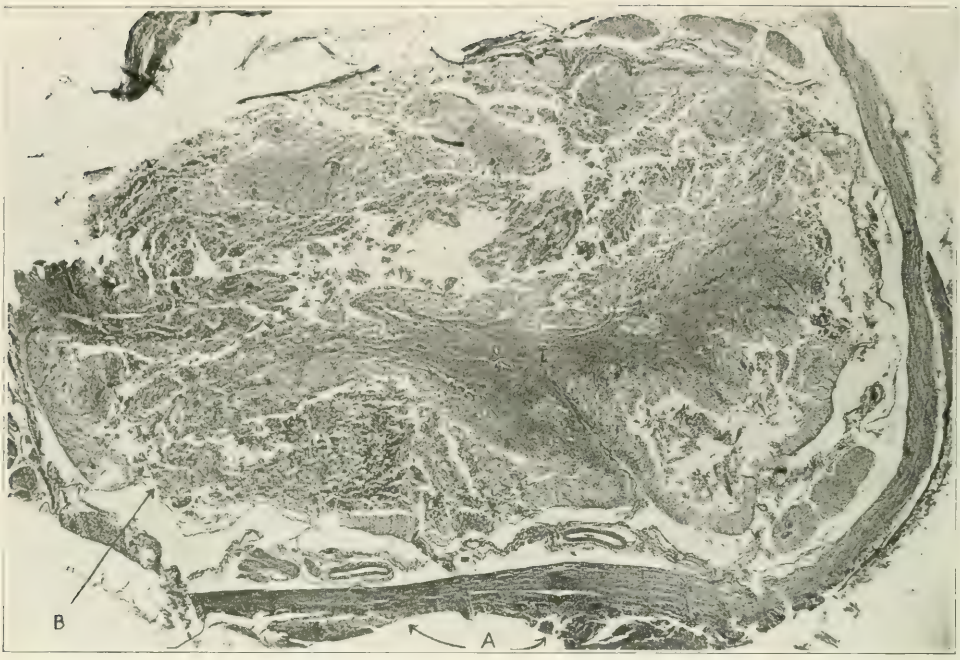

Fig. 1.—Section of cord at ninth dorsal segment; *A*, tumor cells in the dura; *B*, the softened cord.

Fig. 2.—Section of cord at mid-dorsal region: Myelomalacia and cavity formation produced by ascending thrombosis of spinal cord vessels.

to be leukocytes in the spinal fluid. As we have found no inflammatory changes, we feel compelled to believe these cells were not white blood cells but were cells thrown off by the tumor, or possibly gitter cells. Thrombosis of the venous spinal system explains the degeneration above the level of the tumor (Fig. 2) and excuses, perhaps, our erring locali-

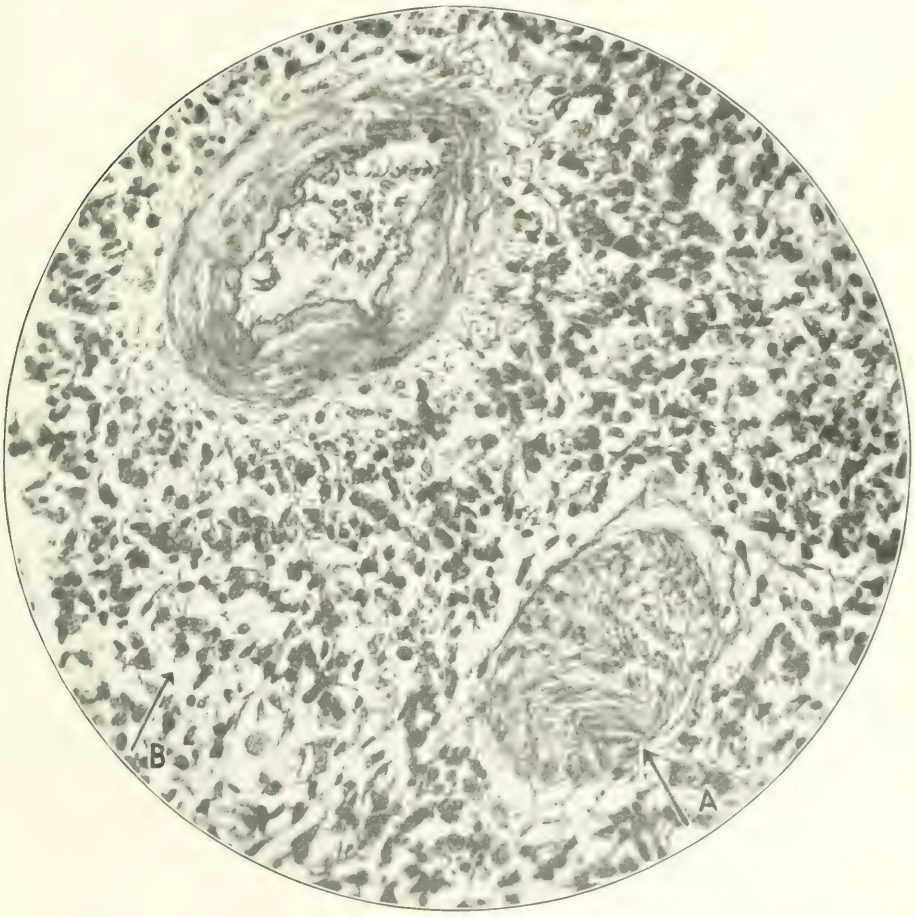

Fig. 3.—Section of liver node; *A*, cross section of adult nerve; *B*, tumor cells.

zation. The original tumor is far from being uncommon, but in metastases we found one feature of the greatest rarity, namely, nerve fibers of adult type (Fig. 3); further, we have been unable to find any case of testicular tumor metastasis to the meninges of the spinal cord (Fig. 4).

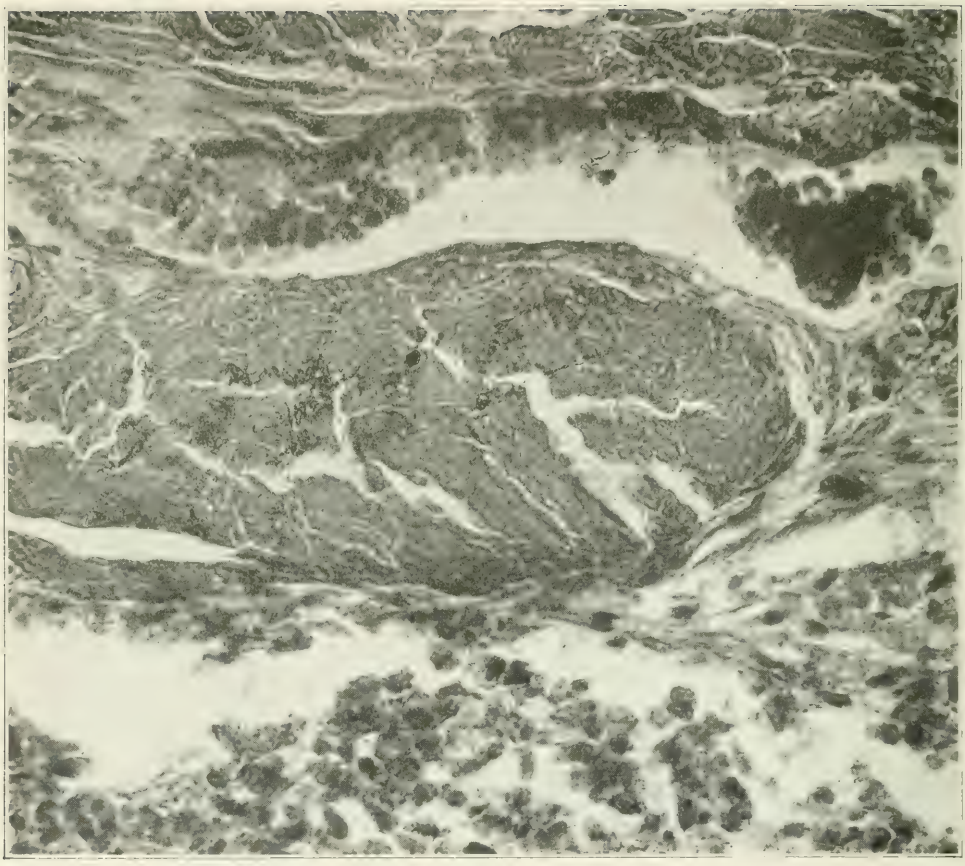

Fig. 4.—A high power magnification of teratomatous theca in which is seen intercalation by tumor cells of the layers of the dura.

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CLINICAL EXPERIMENTS IN MYOTONIA CONGENITA (THOMSEN) WITH ESPECIAL REFERENCE TO THE PARASYMPATHETIC NERVOUS SYSTEM *

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Since Thomsen's [1] description of myotonia congenita and Deleage's [2] observation on atrophy of certain muscle groups in some myotonic patients, a large number of cases of myotonia congenita and myotonia atrophica have been reported. The relationship of these two conditions has been discussed frequently in recent years. The etiology and pathology of myotonia congenita are just as obscure now as they were when this disease was first described.[3, 4]

The observations on the patient described in this paper may throw some light on the nature of this disease and the repetition of these tests on other patients should serve to show whether the conclusions we have drawn can be applied generally to myotonia congenita and myotonia atrophica.

REPORT OF CASE

The patient now under consideration was seen in 1910 by the late Byrom Bramwell [5] who gave a short summary of his history and physical condition. D. F., a book-keeper, aged 32, was admitted to Bellevue Hospital, April 25, 1923, complaining of "contraction of the muscles" and disturbed locomotion. The family history is unimportant, except that one sister suffered from similar disturbances though she is well at present. His past history is negative except for complaints relating to his present condition. The functions of the sensory organs are normal. He suffers from chronic constipation and frequently from palpitation. There is no history of gonorrheal or syphilitic infection. As a child he was always timid and "never cared to talk to people." He is

* From the Department of Neurology of Bellevue Hospital and the Department of Pharmacology of Cornell University Medical College.

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emotional and imaginative. When alone his mind is always active; he sleeps poorly and is tired in the morning. The present trouble began at the age of 9 following "pneumonia of three months' duration." While attending school the patient noticed that "other boys jumped out from the class quicker, and ran up and down stairs faster," than he did. When attempting various movements after rest, tonic spasm of the muscles occurs, preventing, or making difficult, movements of the body, but this disturbance is overcome by effort within from one to two minutes, and ordinary movements are then performed with ease. He is greatly handicapped by his condition. He had a voracious appetite and suffered from severe spastic constipation at the age of 15. He believes that his condition is improved at the present time; his greatest difficulty now being in starting to walk, and in grasping objects. After closing his eyes, tightly, it requires about twenty to thirty seconds before he is able to move the lids. The abdominal muscles contract tonically for ten to fifteen seconds following coughing.

The patient sweats easily; wounds of the body heal slowly; he suffers from headaches, confined to the supraorbital region, at intervals of six or eight weeks, during which there develops ptosis of the left lid with lachrymation, and watery discharge from the left nostril. Through such attacks his muscular disturbance is distinctly improved. The patient also complains of "dizzy spells" which come at irregular intervals and last from one half to one minute in which he practically loses consciousness and following which he develops palpitation for two or three minutes. These attacks of dizziness have no apparent relation to the supra-orbital headaches. The patient's sexual function remained about normal until three years ago, but during the past three years there has been a decrease in desire and last year he lost the sexual function entirely; his weight then decreased by 16 to 20 pounds (7 to 9 kg.).

Condition on Examination.—The patient is a moderately well developed young adult who does not look ill. There is scantily developed hair all over the body with bilateral frontoparietal baldness. The skin is rather smooth and delicate to the touch; the expression of the face is that of a myopathic patient. The medical examination shows normal cranial nerves and normal sensory findings. Examination of the motor system shows that the sternocleidomastoid muscles are rather poorly developed on both sides; the musculature is normally developed in the head, arms and trunk; the muscles of the lower extremities are moderately hypertrophied; no involuntary muscular movements occur. The muscle tone is slightly increased in the arms and markedly increased in the legs.

There are disturbances involving various groups of muscles with actively performed movements. The following muscles show the disturbance to an unusual degree: orbiculares oculorum, orbicularis oris, masseters, pterigoids, flexors of the fingers, opponens of the thumbs, extensors and flexors of the legs and feet, and the abdominal muscles.

Sharp pressure applied to various muscles, including the tongue, produces a "humping" contraction of limited muscular bundles with superficial fibrillatory movements. The coordination of muscles and the gait are normal after the disturbances described above have disappeared. The muscular power of the arms and hands is weak; the maximum compression of the ergometer was 22. The jaw jerk was not elicited; the biceps and triceps jerks were normal; the right lower abdominal reflex was sluggish; the other abdominal regions were not tested; the knee jerks were sluggish, and the ankle jerks were not elicited.

Course.—The patient had two attacks of supra-orbital headache and three "fainting spells" while in hospital. The symptoms of myotonia congenita were

distinctly improved during the period of the left supra-orbital headaches; the drooping of the left eyelid and lachrymation lasted several hours.

Laboratory Findings and Experimental Observations.—Urine: specific gravity 1038; reaction, acid; the tests for glucose, albumin, casts, acetone, diacetic acid, and indican, negative. Blood pressure: 116 systolic, 64 diastolic, and 120 systolic, 70 diastolic at different examinations. Red blood cells: 5,800,000; hemoglobin, over 100 per cent. White blood cells: 12,500; differential count: polymorphonuclear leukocytes, 63 per cent.; lymphocytes, 35 per cent.; transitional cells, 2 per cent.

Blood Chemistry: Nonprotein nitrogen, 35 mg.; creatin, 1.5 mg.; sugar, 95 mg.; calcium, 10 mg. per 100 c.c.; carbon dioxid combining power 42 per cent. Body temperature: from 97.0 to 98.8 F. (36.1 to 36.7 C.). Electrocardiogram (Dr. Eggleston): normal rhythm. Basal metabolism (Miss Hooper): 2,490 c.c. oxygen consumption in ten minutes in two determinations, corresponding to 64 calories per hour, instead of 67 calories, the estimated value, or about 96 per cent. of the normal average.

Roentgen-Ray Examination: There is enlargement of the right hilum nodes, with numerous calcified deposits scattered throughout the right lung; there is enlargement of the right and left auricles, suggesting the presence of mitral valvular disease; no evidence of tuberculosis; the picture of the skull shows a normal sella turcica.

Fluoroscopic Examination: The gastro-intestinal tract is normal except that the proximal portion of the descending duodenum is relatively high and appears to be fixed by adhesions.

Electrical Examination: The order in which contractions were obtained with the galvanic current was: cathodal closure, anodal closure, anodal opening and cathodal opening. More intense threshold current was required to produce faradic contraction of the muscles of the patient than in the case of a normal person. Several muscles showed persistent tetanic contraction after removal of the electrodes. A current which was not sufficient to induce contraction of a muscle group did induce contraction of single muscle bundles with wormlike movements of certain parts of the muscle. The galvanic current at closure induced tetanic contraction of several muscles of the hands and legs. Indirect stimulation of the muscles through the Erb points gave the following threshold intensities of the galvanic current:

	Milliamperes	
Nerves	in this Patient	Normal
Facial	3.5—4.0	1.0—2.5
Median	5.0—6.0	0.3—1.5
Ulnar	4.0—5.0	0.2—0.9
Radial	6.0	0.9—2.7
Peroneal	6.0—7.0	0.9—2.0
Tibial	7.0	0.4—2.0

These results show a marked increase above the normal in the threshold of the galvanic current required in the patient.

Ergographic Observations: Tracings of the movements of the thumbs showed that it required from five to fifteen seconds before the patient was able to respond to the signal with normal contractions. It was observed, however, that the greater the weight employed, the less intense was the muscular disturbance, and when a weight was selected just sufficient to overcome the

spasm of the muscles, the tracing of the muscular contractions was similar to that of a normal individual. This observation shows clearly the independence of the muscular contraction of the muscle tone and it also shows that this disease is due to a disturbance of the muscle tone alone.

Observations on the Autonomic Nervous Sytsem.—As the clinical appearance of the patient made clear several abnormalities in the function of the vegetative nervous system, several tests of the behavior of the autonomic nervous system were made. We do not wish to discuss at this time the value of these tests, but hope to do so later when sufficient data have accumulated.

Bulbar Pressure Test: The normal rhythm was counted three times for periods of ten seconds following pressure on one bulb for a period of ten seconds; repeated tests showed a decrease in the pulse rate of from 6 to 12 per minute.

Vagus Pressure Test: Pressure on one or both of the nerves produced practically no change in the pulse rate.

Vagus Stretching Test ("Höckversuch"): After five successive flexions of the knees, the pulse rate increased by from 5 to 6 beats per minute; during stretching of the vagi, the pulse rate decreased by from 12 to 15 beats per minute, the decrease persisting during one or two minutes.

Influence of Respiration: Deep inspiration caused a decrease in the pulse rate of from 5 to 6 per minute; deep expiration caused an increase of from 9 to 15 per minute.

Epinephrin: A dose of 0.15 mg. of epinephrin in a freshly prepared solution (1:1,000) was injected subcutaneously, after which the changes in blood pressure and pulse rate were observed during a period of sixty-nine minutes. The tracing shows that there was no increase in blood pressure, but, on the contrary, a slight decrease, while there was practically no change in the pulse rate.

Pilocarpin: The subcutaneous injection of 0.15 mg. of pilocarpin hydrochlorid caused practically no change in blood pressure or in the pulse rate, and very little perspiration though the dose was relatively large. Frequent eructation and moderately increased perspiration were the only distinct effects of the drug that were observed.

Atropin: The patient's muscles were tested carefully before the administration of this drug and it was a source of surprise that one hour after the subcutaneous injection of a dose of 0.075 mg. per kilogram of body weight there was a distinct improvement in the myotonic reactions of the muscles. There was complete disappearance of the disturbance of the muscles of the hand, and the muscles of the face showed great improvement, while those of the jaw alone failed to exhibit any change. The muscular disturbance had returned with its usual severity the next morning.

Thyroid Administration: Thyroid substance and thyroxin were administered daily after the observations just described had been made, a total of 105 grains (6.8 gm.) of thyroid substance and 9.6 mg. of thyroxin being given in a period of fourteen days. The patient showed no symptoms of hyperthyroidism although such large amounts were given, but, on the contrary, the muscular contractions became more severe, and the patient became depressed. Within a week he had two attacks of fainting and one of the periodic oculomotor disturbances. The thyroid substance and the thyroxin seemed to aggravate his condition. The tests described above, except that with pilocarpin, were repeated after this thorough thyroidization, and the following results were observed: Pressure on the bulb exerted no influence on the heart rate, but

pressure on the vagi produced the same effect as in the first observations and the influence of the respiration was also the same as that during the earlier tests; stretching the vagi induced a decrease in the heart rate of from 8 to 12 beats, but the slowing was more fleeting than that in the previous tests. Two subcutaneous injections of epinephrin were made. The first of these induced only a slight delayed reaction, while the second caused no change during one hour—the period of observation. Following the subcutaneous injections a dose of 0.5 mg. was injected intravenously without perceptible effect.

The administration of atropin was then repeated following an unusually severe muscular disturbance, during which seven seconds were required to open the eyes after their voluntary firm closure, and seven or eight efforts were made to open the mouth when it was firmly closed. The muscular disturbance in the hand was so severe that the patient was unable to relax the fingers for several seconds after having grasped an object. The same dose of atropin was administered in the same dilution as previously (0.075 mg. per kilogram of body weight, 6 mg. total). The effect of this dose was surprising. The pulse rate increased gradually, nine minutes after the injection, but the blood pressure showed no change for fifty-four minutes when the patient suddenly complained of feeling sick; at the same time there was a marked increase in the blood pressure with an increase in the heart rate. Twenty minutes after the injection of atropin (subsequent to thyroidization) there was a marked improvement in the muscular disturbance, and after an hour the patient was able to make every muscular movement with normal ease and speed. An hour later the test of the muscles was repeated. *The myotonic reaction of the muscles, including the fibrillary contractions of the muscle bundles after mechanical stimuli, were completely absent.*

The patient complained of "fainting spells" and of the presence of a peculiar odor. He was rather apprehensive but stated that the muscles felt much "lighter." The following morning the patient reported that he felt normal except for blurred vision. At that time the myotonic reaction of the muscles was slight. He stated that he was able to flex the lower extremities in certain directions as he had never been able to do before. Two days later the muscular disturbance was of its usual severity, and the patient complained of palpitation and refused to take atropin. When discharged from the hospital the patient was advised to take atropin three or four times daily in doses of $\frac{1}{100}$ grain (0.0006 gm.) until beneficial effects developed. The patient has been seen once since his discharge from the hospital at which time he reported slight improvement, which he attributed to the use of atropin, though he complained of his inability to take atropin regularly because it disturbed his cardiovascular system.

COMMENT

This patient exhibits several interesting clinical features which illustrate the difficulties encountered in the differentiation of myotonia congenita (Thomsen) from myotonia atrophica (Deleage). The history goes back to early childhood, and though the patient manifests many signs of myotonia congenita,[6] he shows atrophy of certain muscles, general muscular weakness, and psychic and internal secretory disturbances,

6. Strümpell: Spezielle Pathologie und Therapie der inneren Krankheiten, Leipzig, 1918.

absence of certain reflexes—signs often described as characteristic of myotonia atrophica. Orschowsky [7] is the only observer who states that there is an element of muscular weakness and atrophy in every case of myotonia congenita.

We have no evidence in this case that the pathologic condition is localized in the muscles. We did not perform a biopsy, but the patient stated that it had been performed on him three times and that no histologic changes in the structure of the muscles were observed. We were unable to obtain the previous hospital charts of this patient owing to their having been accidentally destroyed. The blood chemistry and

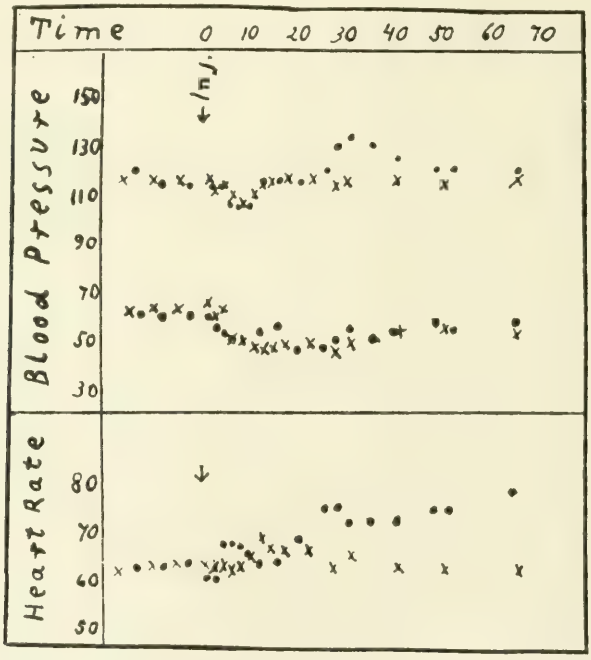

Chart 1.—The effect of epinephrin (1.0 mg. total) on the blood pressure and heart rate, before and after taking thyroid. The crosses indicate the findings before, and the dots the findings after thyroidization.

basal metabolism were normal. One of us [8] has recorded observations on a patient suffering with myotonia congenita in which case P. Hari made metabolic determinations during rest and muscular exercise. The

7. Orzechowsky: La componante parétique du trouble myotonique, Bull. de l'Acad. polon. des science méd. **1**:1, 1921; Zentralbl. f. ges. Neurol. u. Psychiat. **28**:357, 1922.

8. Weiss, S.: Ueber die Bedeutung des erhöhten respiratorischen Quotienten bei forcierter Athmung und erhöhter Muskelarbeit, Biochem. Ztschr. **101**:7, 1920.

results indicate no essential difference in the metabolism of that patient from that of a normal individual, though one would anticipate a marked difference if the disease were primarily of muscular origin.

The patient did not react to epinephrin either before or after the administration of thyroid substance, but he showed marked improvement from the use of atropin, especially after thyroidization. The entire experiment, especially (*a*) the disappearance of myoclonic reactions in affected muscles for twelve hours after the injection of atropin and thyroid, (*b*) the lack of reaction to epinephrin, and (*c*) the fact that atropin (a specific depressant to the parasympathetic nervous system) caused the return of normal muscle function, suggests strongly an abnormal increase in the activity of the parasympathetic nervous system.

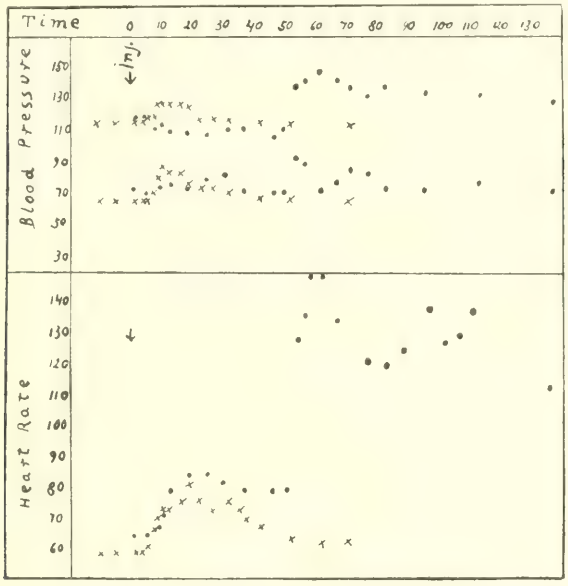

Chart 2.—The effect of atropin (6.0 mg. total) on the heart rate and blood pressure before and after taking thyroid. The crosses indicate the findings before, and the dots the findings after thyroidization.

The oculocardiac disturbance, the slow normal pulse, and the spastic constipation point in the same direction. The periodic disturbance in the function of the oculomotor nerves (parasympathetic in behavior) must be looked on also as a sign of disturbed parasympathetic function. This is the more important because during the past few years the independence of muscle tone and muscular contraction has been proved.[9]

9. Hunt, J. R.: The Dual Nature of the Efferent Nervous System, Arch. Neurol. & Psychiat. **10**:37 (July) 1923.

Evidence accumulates that muscle tone is maintained by the autonomic nervous system and very probably by the parasympathetic division.[10] It is suggested, therefore, that the pathologic condition in this case depends on a disturbance of muscular tone itself due to abnormalities of the parasympathetic nervous system.

The great confusion in our knowledge of the pathology of myotonia congenita may be due to the fact that the reported cases have been studied morphologically and with little attention to the functional pathology of the disease.

CONCLUSIONS

These observations afford no evidence that the disease is primarily of muscular origin, but they point toward the fact that it is due to an abnormally increased activity of the parasympathetic nervous system which normally regulates the tone of striated muscles.

The administration of thyroid substance and thyroxin exerted the same sensitizing action on the effect of atropin in this case that they exert on the effect of epinephrin in normal individuals This suggests that the thyroid secretion is a sensitizing agent for both the sympathetic and the parasympathetic systems.

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10. Spiegel, E. A.: Zur Physiologie und Pathologie des Skelettmuskeltonus, Ztschr. f. Neurol. u. Psychiat. **81**:517, 1923.

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A NOTE ON THE RELATION OF THE AXILLARY ARTERY TO THE BRACHIAL PLEXUS

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A NOTE ON THE RELATION OF THE AXILLARY ARTERY TO THE BRACHIAL PLEXUS

By Walter M. Kraus, A.M., M.D.

NEW YORK

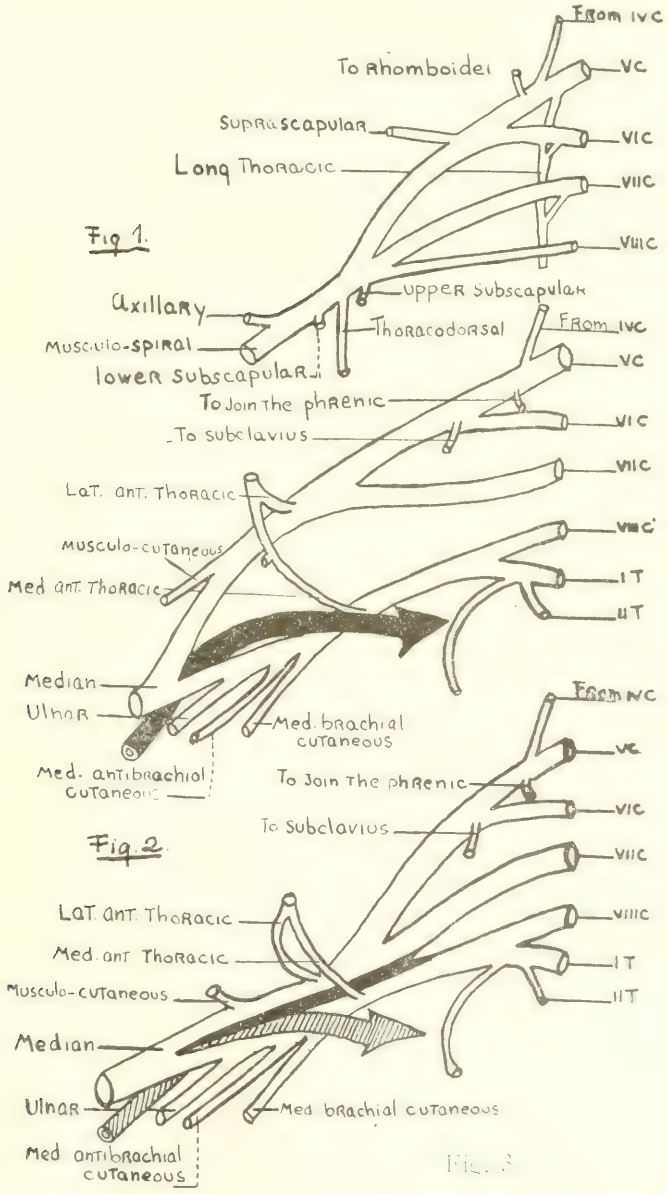
A. *Introduction.*—The brachial plexus is not a very simple structure. However, the use of a bit of anatomical information may serve to make its complicated branching very much easier to remember.

The division into dorsal and ventral branches.—The first fact of importance is that it is composed of branches which may be divided into ventral and dorsal groups. These ventral and dorsal branches arise in the ventral branches of spinal nerves. If, for the sake of simplicity only the five main branches which go to the muscles and skin of the arm, forearm, hand and fingers are considered, we find that these may be divided into two general groups, the circumflex and musculospiral on one hand, the musculocutaneous, median and ulnar on the other. If we represent these two groups separately we get pictures which are shown in Figures 1 and 2.*

B. *The part played by the axillary artery.*—Figure 3 shows a more complicated interlacing. This is readily understood, however, by the second anatomical fact which is the one to be emphasized. The axillary artery is at the bottom of the complexity. If we assume that the axillary artery were differently placed than it is, that it did not pass between the two heads which make up the median nerve (Fig. 3) it will be seen that the ventral nerve of the arm, forearm, hand and fingers, that is, the combined outer and inner cord, would be fused and as simply constituted as the dorsal nerve, the posterior cord. The direction of fusion of nerves is always towards the axis of the central nervous system, just as the direction of branching is always away from it. It is quite obvious therefore, that the presence of the axillary artery accounts for the lack of fusion and hence the complexity. The musculocutaneous, median and

* Note.—The reader will find the following an excellent article on the normal and abnormal structure of the brachial plexus: "The Brachial Plexus of Nerves in Man—the Variations in Its Formation and Branches." Abram T. Kerr. The American Jr. of Anatomy, Vol. 23, No. 2, March 15, 1918, pp. 285-395.

ulnar nerves are but branches of the ventral nerve dignified by special names. The circumflex and musculospiral nerves are but



branches of the dorsal nerve also dignified by separate names. The reason that these branches occur is largely due to the necessity of

supplying *topographically distant* structures, that is, the muscles of the shoulder on one hand and the muscles of the arm and forearm on the other.

KEY TO DIAGRAM (p. 323)

Figure 1 represents the dorsal trunks and branches of the brachial plexus. The fusion into a single branch, the posterior cord, is shown.

Figure 2 represents the ventral trunks and branches of the brachial plexus. The axillary artery is represented by the black arrow. The way in which it prevents fusion of the two heads of the median is illustrated by the course of the artery.

Figure 3 is schematic and shows what would happen if the axillary artery were absent. The black area between the upper and lower cords indicates their fusion, which would occur if the artery were not present. These two cords would then form a single ventral cord like the dorsal or posterior cord illustrated in figure 1.

The diagram illustrates the principle that the direction of fusion of nerves is always towards the axis of the central nervous system while the direction of branching is always away from it. These two activities are constantly at work. As a result, extremely complex internal nerve plexuses are produced.

The position of the axillary artery represents one of the many anatomical deterrents of fusion centralward of peripheral nerves.

An Interpretation of the Posture of Parkinsonian Syndromes in Terms of the Neuro-Muscular Mechanism

BY

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AN INTERPRETATION OF THE POSTURE OF PARKINSONIAN SYNDROMES IN TERMS OF THE NEUROMUSCULAR MECHANISM.

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I. The Posture of the Parkinsonian Syndrome

At the present time the positions of the various parts of the body in the Parkinsonian syndrome, when described only in terms of muscle movements, that is, of flexion, extension, etc., give no notion of uniformity, no notion of a homogeneous reaction due to disease of the nervous system. The position of the fingers illustrates this very clearly. They are usually flexed at the metacarpo-phalangeal joints and extended at the interphalangeal joints. Gowers has called this the interosseal position. As to the rest of the body, it may be said in general that the arms are held flexed and adducted, that in severe cases the neck and trunk are flexed, and that there is flexion at the hip and at the knees. In the rare instances where contracture affects the feet, extension occurs, producing a pes equinus or equino-varus. In the forms of the disease due to arteriosclerosis involvement of the feet is not common. However, in those forms due to epidemic encephalitis a pes equinus or pes equino-varus is often found.

Certainly it is difficult to interpret such a mixture of flexion, extension, etc., as indicative of a uniform reaction of the nervous system or of the muscular system due to disease. Nor do we find this so expressed in the present descriptions. However, if movement is described in terms of the neuromuscular instead of the muscular mechanism, that is, if we take into consideration peripheral motor neurones as innervators of muscles, the grouping of these neurones and their control by the spinal cord as well as the embryology and grouping of muscles, we find that the picture is greatly simplified.

Both the nerves and muscles of the trunk and extremities are divisible into a large dorsal and a large ventral group. (Tables 1, 2, 3, 4.) In the Parkinsonian syndrome it is very easy to show that the ventral group becomes more active than the dorsal group. An imbalance exists between these two large groups in spite of the fact that no paralysis of voluntary power is present. This imbalance is due to abnormal innervation of antagonistic groups of muscles.

The nervous system surely is so constructed that it may produce various muscular patterns. These patterns or positions are due to variations in the innervation of antagonistic groups of muscles. For example, in post-hemiplegic contracture of the arm, there is flexion at the elbow. The biceps is more powerfully innervated than the triceps. In decerebrate rigidity there is extension at the elbow. The opposite conditions obtain. The triceps is more powerfully innervated than the biceps. These patterns are constants.

The force which activates muscles in these patterns is tonus. For example, under certain experimental or clinical conditions, the muscles of the body assume a pattern which has been termed decerebrate rigidity by S. A. K. Wilson[1] and which was previously designated by Sherrington[2] as the antigravity position. So long as tonus exists this position exists. When tonus is eliminated the position disappears. In the Parkinsonian syndrome abnormalities of tone usually exist. There is never a complete *absence* of tone, flaccidity. Tone is sometimes normal, usually increased. There is a second factor. The pathological process has liberated parts of the nervous system capable of producing an abnormal posture or muscular pattern due to abnormality of innervation of antagonistic groups of muscles. Into this pattern tone flows. The activity of hyoscine in diminishing the severity of this abnormal posture or pattern by reducing tone forms a proof of the explanation given.

TABLE I.

Division of the Nerves of the Extremities Into Ventral and Dorsal Groups

Origin		Nerves
Upper Extremity		
Brachial Plexus	Dorsal trunks (Posterior cord)	Dorsal scapular Long thoracic Suprascapular Subscapular (2) Thoraco-dorsal Axillary Radial
	Ventral trunks (Lateral and medial cords)	Nerve to subclavius Anterior thoracic (2) Musculo-cutaneous Median Ulnar
Lower Extremity		
Lumbo-Sacral Plexus	Dorsal trunks	Superior gluteal Inferior gluteal Nerve to piriformis Femoral Peroneal
	Ventral trunks	Obturator Nerve to obturator internus and superior gemellus Nerve to quadratus femoris and inferior gemellus Tibial

TABLE II.

Axial Muscles

Muscles of the Back	*Ventral*	*Dorsal*
Serratus posterior superior		x
Serratus posterior inferior		x
Splenius		x
Sacrospinalis		x
Iliocostalis		x
Longissimus		x
Spinalis dorsi		x
Semispinalis		x
Multifidus		x
Obliquus capitis inferior		x
Obliquus capitis superior		x
Rectus capitis posterior major		x
Rectus capitis posterior minor		x
Rotatores		x
Interspinales		x
Intertransversarii	x	
Trapezius		x
uscles of the Neck		
Scalenus anterior	x	
Scalenus medius	x	
Scalenus posterior	x	
Longus capitis	x	
Rectus capitis anterior	x	
Longus colli	x	
Rectus capitis lateralis	x	
Sterno-kleido-mastoid	x	
Muscles of the Thorax		
Intercostales	x	
Levatores costarum	x	
Subcostales	x	
Transversus thoracis	x	
Muscles of the Abdominal Wall		
Obliquus externus abdominis	x	
Obliquus internus abdominis	x	
Cremaster	x	
Transversus abdominis	x	
Pyramidalis abdominis	x	
Rectus abdominis	x	
Quadratus lumborum	x	
uscles of the Perineum		
Sphincter ani externus	x	
Corrugator cutis ani	x	
Transversus perinei superficialis	x	
Bulbocavernosus	x	
Ischiocavernosus	x	
Sphincter urethrae membranaceae	x	
Transversus perinei profundus	x	
Muscles of the Pelvis		
Levator ani	x	
Coccygeus	x	

TABLE III.

Muscles of the Upper Extremity.

Dorsal	*Ventral*
1. Levator scapulae	
2. Serratus anterior	
3. Rhomboideus major	
4. Rhomboideus minor	
5. Supraspinatus	
6. Infraspinatus	
7. Teres minor	
8. Deltoid	
9. Subscapularis	
10. Teres major	
11. Latissimus dorsi	
	12. Subclavius
	13. Pectoralis major
	14. Pectoralis minor
	15. Biceps brachii
	16. Brachialis*
	17. Coracobrachialis
18. Brachio-radialis	
19. Extensor carpi radialis longus	
20. Extensor carpi radialis brevis	
21. Supinator	
22. Extensor pollicis longus	
23. Extensor indicis proprius	
24. Abductor pollicis longus	
25. Extensor pollicis brevis	
26. Extensor communis digitorum	
27. Extensor carpi ulnaris	
28. Extensor minimi digiti quinti	
29. Anconeus	
30. Triceps	
	31. Pronator teres
	32. Flexor carpi radialis
	33. Palmaris longus
	34. Flexor digitorum sublimis
	35. Flexor digitorum profundus
	36. Flexor pollicis longus
	37. Pronator quadratus
	38. Lumbrical 1
	39. Lumbrical 2
	40. Lumbrical 3
	41. Lumbrical 4
	42. Abductor pollicis brevis
	43. Opponens pollicis
	44. Flexor pollicis brevis. Lateral head
	45. Flexor pollicis brevis. Medial head
	46. Flexor carpi ulnaris
	47. Adductor pollicis obliquus
	48. Adductor pollicis transversus.
	49. Interosseus volaris 1
	50. Interosseus volaris 2
	51. Interosseus volaris 3
	52. Interosseus dorsalis 1
	53. Interosseus dorsalis 2
	54. Interosseus dorsalis 3
	55. Interosseus dorsalis 4
	56. Opponens digiti quinti
	57. Flexor digiti quinti brevis
	58. Abductor digiti quinti

* Supplied by both ventral and dorsal nerves.

TABLE IV.

MUSCLES OF THE LOWER EXTREMITY.

Dorsal	*Ventral*
1. Iliacus	
2. Psoas major	
3. Psoas minor	
4. Pectineus*	
5. Sartorius	
6. Rectus femoris	
7. Vastus lateralis	
8. Vastus medialis	
9. Vastus intermedius	
10. Tensor fasciae latae	
11. Glutaeus minimus	
12. Glutaeus medius	
13. Piriformis	
14. Glutaeus maximus	
15. Biceps femoris (shorthead)	16. Biceps femoris (longhead)
	17. Semitendinosus
	18. Semimembranosus
	19. Adductor magnus
	20. Obturator externus
	21. Adductor longus
	22. Adductor brevis
	23. Gracilis
	24. Obturator internus
	25. Superior gemellus
	26. Inferior gemellus
	27. Quadratus femoris
28. Tibialis anterior	
29. Extensor hallucis longus	
30. Extensor digitorum longus	
31. Peronaeus tertius	
32. Peronaeus longus	
33. Peronaeus brevis	
34. Extensor digitorum brevis	
	35. Gastrocnemius
	36. Soleus
	37. Plantaris
	38. Popliteus
	39. Tibialis posterior
	40. Flexor digitorum longus
	41. Flexor hallucis longus
	42. Quadratus plantae
	43. Abductor digiti quinti
	44. Flexor digiti quinti brevis
	45. Opponens digiti quinti
	46. Plantar interosseus 1
	47. Plantar interosseus 2
	48. Plantar interosseus 3
	49. Dorsal interosseus 1
	50. Dorsal interosseus 2
	51. Dorsal interosseus 3
	52. Dorsal interosseus 4
	53. Adductor hallucis
	54. Lumbrical 1
	55. Lumbrical 2
	56. Lumbrical 3
	57. Lumbrical 4
	58. Abductor hallucis
	59. Flexor digitorum brevis
	60. Flexor hallucis brevis

* Supplied by both ventral and dorsal nerves.

In order to establish the muscular pattern it is necessary to consider the position of the various parts of the body in both unilateral and bilateral Parkinsonian syndromes.

A. The Position of the Hand

In the Parkinsonian syndrome the fingers are usually extended at the phalangeal and flexed at the matacarpo-phalangeal joints. The thumb is slightly adducted, certainly not extended. This position is entirely dependent upon the activities of the ventral intrinsic muscles of the hand. All of these muscles are innervated by the ventral median and ulnar nerves. (Table 3.) Were the dorsal muscles controlling the fingers at all hyperactive we should have a position approaching that of a clawhand which is indeed opposite to that which occurs in the Parkinsonian syndrome.

When the postural defect is more exaggerated, the long flexors of the thumb and fingers bring them into a position of flexion and the hand slowly assumes the classical position found in the hypertonic type of hemiplegia. The remainder of the arm is flexed at the wrist and elbow joints and adducted at the shoulder—all evidence of hyperactivity of ventrally innervated muscles which have developed on the ventral aspect of the limb.

The extension of the fingers at the interphalangeal joints by the interossei is of ventral origin and may be spoken of as ventral extension in order to make clear not only the functions of the muscles concerned ("extension") but also their innervation and their embryology ("ventral"). Thus it is seen that the entire reaction of the upper extremities is but a manifestation of an imbalance between the activation of ventral and dorsal muscles in which the ventral group dominates the picture.

B. The Head

When both arms are affected the head is flexed and a certain degree of flexion appears in the upper portions of the trunk, based upon a close

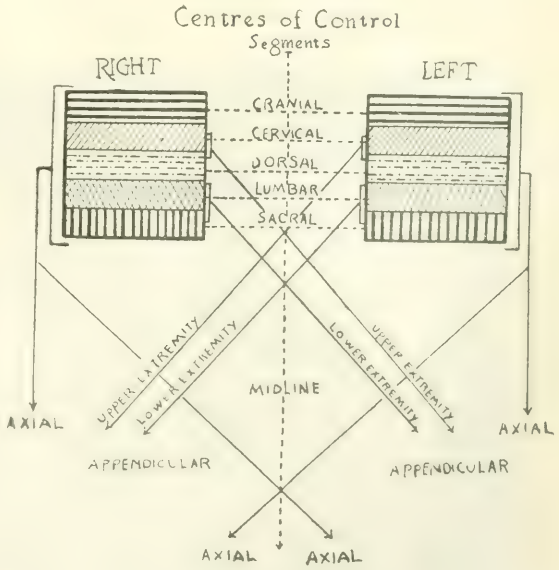

FIG. 1

anatomical relationship of the centres of control of the muscles of the upper trunk and arms. In the re-representation at higher levels of the seg-

mental relations in the spinal cord, the same topographical relations must obtain (Fig. 1). It is but natural therefore, that, when both arms become affected the upper portions of the trunk should also be involved.

The diagram (Fig. 1) illustrates a center controlling ventral and dorsal movements. This diagram represents the right extremity, for example, innervated by one side only of the nervous system, while the same side innervates both halves of the axial musculature. A lesion may affect the centers of one side without affecting the axial musculature, since this is also supplied by the other side. The moment, however, that the lesion becomes bilateral, both unilaterally innervated arms and the innervation of the axial muscles become affected, thus producing the bending or flexion of the head and trunk. This diagram illustrates not only how asymmetrical types occur but also the basis for the common cranio-cervico-brachial types involving the head, neck and arms and the relatively rare lumbosacral types involving the lower extremities.

C. The Trunk and Leg

In strictly unilateral Parkinsonian syndromes the patient maintains a normally erect position. Neither the hip nor the trunk are flexed. As soon as the disease becomes bilateral, flexion appears, first of the head. It may spread to the trunk and hips. Flexion of the head and of the main part of the trunk is obviously due to a relative over-activity of the ventral musculature as compared to that of the dorsal. It is more difficult to explain flexion at the hip. This movement is carried out mainly by the dorsal ileopsoas, sartorius and tensor fasciae latae and may be called dorsal flexion. However, the condition in unilateral syndromes throws light upon this. In these cases there is no flexion at the hip. The dorsal flexor group is not active. If it were affected and were the cause of the flexion at the hip, it would be involved in the unilateral types, since its innervation is unilateral like that of the appendicular musculature of which it forms a part, and not bilateral as is that of the axial musculature. From this it may be seen that the movement of flexion at the hip has not been brought about by the dorsal ileopsoas, sartorius and tensor fasciae latae but is due to the activity of ventral trunk muscles.

The flexion at the knee is a compensatory response to the pushing forward of the center of gravity, brought about by the overactivity of the ventral trunk muscles.

When the Parkinsonian walks or runs he shows a more or less pronounced tendency to rise upon his toes, which is a manifestation of overactivity of the ventral muscles in the back of the calf.

D. Cases Showing Asymmetrical Involvement

In this group of cases in which the involvement is more extensive on one side of the brain than on the other, the notion that the postural defect of Parkinson's disease is due to an imbalance of ventral and dorsal muscles in which the ventral group dominates, is very clearly emphasized. Figures 2, 3 and 4 represent two different types of Parkinsonian syndrome. The first two are

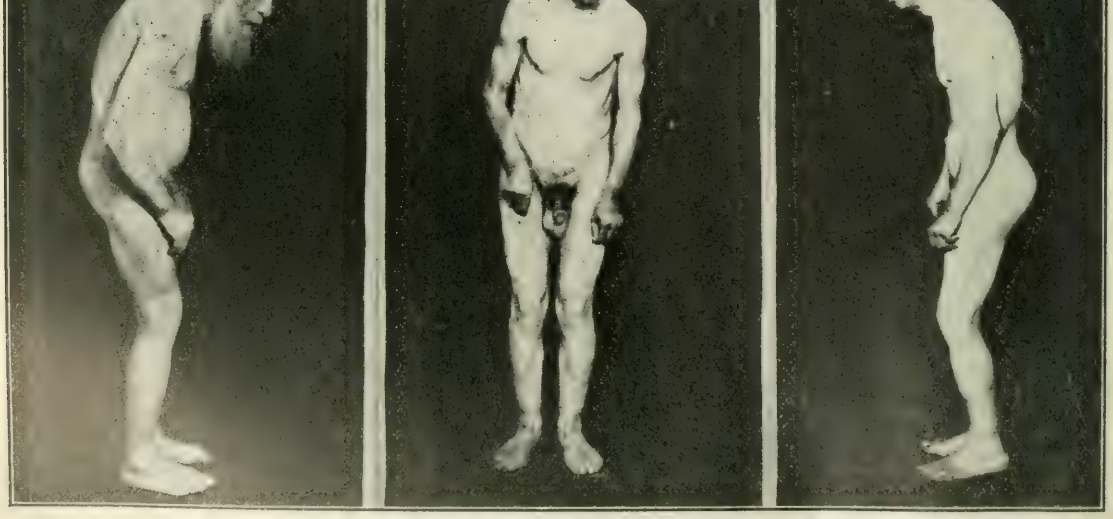

FIG. 2

FIG. 3—Note the greater involvement of the right sterno-kleido-mastoid and of the muscles of the right arm.

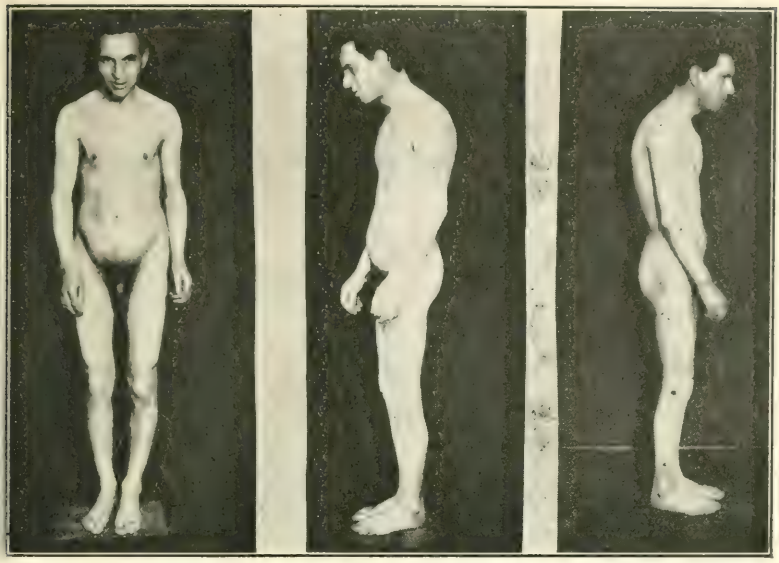

Fig. 4—Note the greater involvement of the right arm.

lue to arteriosclerosis, the last due to epidemic encephalitis. In figure 3 a greater involvement on the right side is clearly seen in the arm, in which flexion at the elbow and fingers is more pronounced. The front view indicates also that he right sterno-kleido-mastoid is more active han the left, which brings the head around from he mid-position. The sterno-kleido-mastoid is a ventral muscle. In Figure 4 it will be seen from he front view that the right arm is more affected han the left. The comparison of the positions of the right and left hand in the lateral views emphasizes this difference. In asymmetric cases herefore, the side showing the greater involvenent shows a greater activity of ventral muscles. A few words will suffice to indicate that the pes equinus and flexion of the toes, as well as the occasional varus, present like the other positions of the Parkinsonian syndrome, one due to over-activity of ventral muscles. All of the intrinsic muscles of the foot are like those of the hand, ventral. The carrying down of the foot is due to the action of the ventral gastrocnemii and soleus.

It is therefore apparent that the posture of a Parkinsonian syndrome represents an imbalance in the innervation of some or all of the groups of muscles of the trunk and extremities, from which there results a pattern indicative of an overactivity of the ventral group.

The involvement is, of course, not always complete either on one side or symmetrical or complete on both sides, depending upon the extent of actual anatomical involvement. Since the re-representation at higher levels must be similar to that of the segmental arrangements of the spinal cord, it is but natural that in the vast majority of cases, adjacent centers, such as the head and upper extremities, will be simultaneously involved.

II. Contractures in the Parkinsonian Syndrome

The occurrence of contractures in the Parkinsonian syndrome is rare. They are more rare in the vascular type of the disease than in those following epidemic encephalitis. Dejerine states

Fig. 5

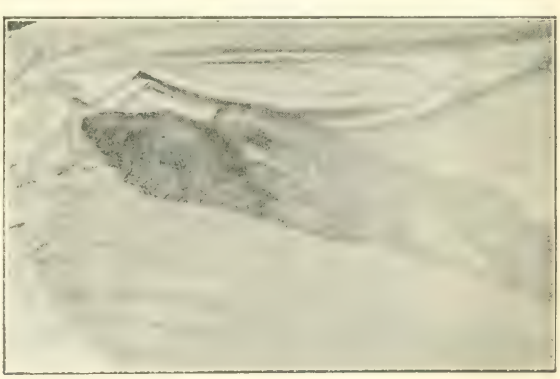

Fig. 5

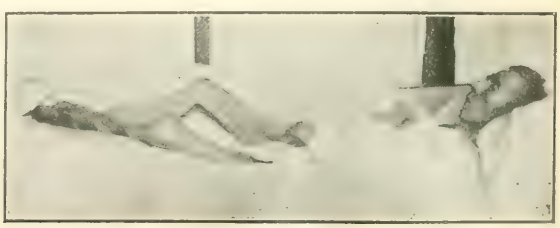

Fig. 5

"this abnormality of the foot in Parkinson's disease (which to my knowledge has not been reported) is very rare and I have up to now, observed only two cases."[3] Though I have seen many cases of the Parkinsonian syndrome I recall seeing but three showing contracture of the feet, when the cause of the disease was other than epidemic encephalitis. Figure 5 shows a very severe contracture. These contractures are due to overactivity of ventral muscles and form additional evidence of the thesis put forward in this article. The rarity of contractures indicates that disease of some region other than that causing the usual picture of the Parkinsonian syndrome has appeared.

III. Conclusions

1. The defects in posture and the contractures of the Parkinsonian syndrome indicate an imbalance between the activities of certain ventral and dorsal muscles. As a result of this imbalance the activity of certain ventral muscles becomes greater than that of certain dorsal muscles.

2. Normal or, better, increased tone, is required to make this imbalance manifest.

3. The defects of posture and of tone are separate and distinct and are not due to disorder of a single physiological system.

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Note: I wish to express my thanks to Drs. S. P. Goodhart and Foster Kennedy for permission to use photographs of cases on their respective services.

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Original Papers.

ON THE PRODUCTION OF NEUROMUSCULAR PATTERNS BY RELEASE OF SPINAL INTEGRATIONS AFTER DECEREBRATION.*

BY WALTER M. KRAUS AND ABRAHAM M. RABINER, NEW YORK.

I.—INTRODUCTION.
II.—THE NEUROMUSCULAR MECHANISM.
III.—A NEUROMUSCULAR ANALYSIS OF THE EXPERIMENTAL AND CLINICAL EVIDENCE FOR DECEREBRATE RIGIDITY AND THE FLEXION REFLEX.
A. The Experimental Evidence.
B. The Clinical Evidence for Decerebrate Rigidity.
C. The Clinical Evidence for the Flexion Reflex.
D. Summary.
IV.—CASE REPORTS.
V.—THE FLEXION REFLEX POSITION OF THE ARM.
VI.—THE RELATION OF POSTURE PATTERNS TO TONE.
VII.—THE KINETIC AND STATIC TYPES.
VIII.—SUMMARY AND CONCLUSIONS.

I.—INTRODUCTION.

THANKS to the efforts of S. A. K. Wilson, we now have a clinical application of that part of the experimental work of Sherrington which demonstrated the existence, after removal of the more anterior parts of the central nervous system, of the condition known as decerebrate rigidity. This clinical and pathological study promises to clear up many of the obscure problems, not only of defects of posture, but of the physiology of the nervous system in general. It

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is our desire to assist in this by analyzing, from a new angle, a series of cases showing decerebrate posture.

One of us[1] in November, 1921, presented a preliminary general outline of this new approach to the subject of motility and posture. In this paper decerebrate postures will be considered from that point of view.

II.—THE NEUROMUSCULAR MECHANISM.

The essential point of the neuromuscular approach to the subject of motility and posture lies in two assumptions. One is that a description of groups of movements in terms indicating merely the activity of muscles in changing the position of various parts of the body in a geometrical sense has, on careful analysis, no definite relationship to any integration of movements by the spinal cord or higher centres of the nervous system. The other assumption is that movements, grouped or isolated, must be considered as activated by the nervous system in patterns primarily dependent upon the primitive and anatomical grouping of muscles, and not upon their function only. The peripheral motor neurones and their end-organs, the muscles, have group relations indicating integration by the spinal cord. This implies that the anatomical grouping of muscles, the common nerve-supply of certain groups of them, their common origin on certain aspects of the body, must form bases for the interpretation of group movements. The simplest example of this is the division of the musculature of the back, neck, and abdominal wall into dorsal and ventrolateral groups. The application of this information to physiology reveals immediately that the dorsal group causes extension while the ventrolateral group causes flexion. The lateral and rotatory movements produced by these groups of muscles are due to over-activity of the right or left halves of either the dorsal or ventral groups or both.

In the more distant portions of the extremities the muscles also develop in large 'premuscle masses' from the ventral and dorsal aspects.[2] These masses give rise to groups of muscles whose nerve-supply are respectively dorsal and ventral. For example, of the branches of the brachial plexus going to the arm, forearm, and hand, the circumflex and musculospiral are dorsal, and innervate muscles derived from the dorsal premuscle mass, while the musculocutaneous, median, and ulnar are ventral, and supply muscles derived from the ventral premuscle mass (*see Table IV*). However, an examination of the functions of muscles does not always reveal a correspondence between actual function and that expected. In other words, muscles developing on the dorsal aspect of the limb have not always dorsal functions, such as extension, and vice versa. The best examples of

this are as follows: The muscles iliopsoas, pectineus, sartorius, on the anterior aspect of the thigh, since they are dorsal in origin (part of the so-called femoral group), would, by analogy with the axial muscles, be expected to have an extensor function, whereas, in reality, their function is flexor. In the hand the interossei extend the terminal phalanges, though, with the exception of the abductor digiti quinti, all the other muscles supplied by the ulnar nerve and of the same group have a flexor function. Since the actual anatomical facts form the basis of the entire matter, these must be presented at once. In *Tables I, II, and III* the muscles of the body, except those supplied by cranial nerves, will be found arranged in dorsal and ventral groups. In *Table IV* the spinal motor nerves will be found so arranged. This last tabulation has been taken from Patterson's article in Cunningham's *Text-book of Anatomy*.[3]

Table I.—Division of the Muscles of the Upper Extremity into Ventral and Dorsal Groups.

Muscle	Ventral	Dorsal
1.—Levator scapulæ		×
2.—Serratus anterior		×
3.—Rhomboideus major		×
4.—Rhomboideus minor		×
5.—Supraspinatus		×
6.—Infraspinatus		×
7.—Teres minor		×
8.—Deltoid		×
9.—Subscapularis		×
10.—Teres major		×
11.—Latissimus dorsi		×
12.—Subclavius	×	
13.—Pectoralis major	×	
14.—Pectoralis minor	×	
15.—Biceps brachii	×	
16.—Brachialis*	×	
17.—Coracobrachialis	×	
18.—Brachioradialis		×
19.—Extensor carpi radialis longus		×
20.—Extensor carpi radialis brevis		×
21.—Supinator		×
22.—Extensor pollicis longus		×
23.—Extensor indicis proprius		×
24.—Abductor pollicis longus		×
25.—Extensor pollicis brevis		×
26.—Extensor communis digitorum		×
27.—Extensor carpi ulnaris		×
28.—Extensor minimi digiti quinti		×
29.—Anconeus		×
30.—Triceps		×

*Supplied by both dorsal and ventral nerves.

Table I.—Division of the Muscles of the Upper Extremity into Ventral and Dorsal Groups—*continued.*

Muscle	Ventral	Dorsal
31.—Pronator teres	×	
32.—Flexor carpi radialis	×	
33.—Palmaris longus	×	
34.—Flexor digitorum sublimis	×	
35.—Flexor digitorum profundus	×	
36.—Flexor pollicis longus	×	
37.—Pronator quadratus	×	
38.—Lumbricalis 1	×	
39.—Lumbricalis 2	×	
40.—Lumbricalis 3	×	
41.—Lumbricalis 4	×	
42.—Abductor pollicis brevis	×	
43.—Opponens pollicis	×	
44.—Flexor pollicis brevis (lateral head)	×	
45.—Flexor pollicis brevis (medial head)	×	
46.—Flexor carpi ulnaris	×	
47.—Adductor pollicis obliquus	×	
48.—Adductor pollicis transversus	×	
49.—Interosseus volaris 1	×	
50.—Interosseus volaris 2	×	
51.—Interosseus volaris 3	×	
52.—Interosseus dorsalis 1	×	
53.—Interosseus dorsalis 2	×	
54.—Interosseus dorsalis 3	×	
55.—Interosseus dorsalis 4	×	
56.—Opponens digiti quinti	×	
57.—Flexor digiti quinti brevis	×	
58.—Abductor digiti quinti	×	

Table II.—Division of the Muscles of the Lower Extremity into Ventral and Dorsal Groups.

Muscle	Ventral	Dorsal
1.—Iliacus		×
2.—Psoas major		×
3.—Psoas minor		×
4.—Pectineus*		×
5.—Sartorius		×
6.—Rectus femoris		×
7.—Vastus lateralis		×
8.—Vastus medialis		×
9.—Vastus intermedius		×

*Supplied by both dorsal and ventral nerves.

Table II.—Division of the Muscles of the Lower Extremity into Ventral and Dorsal Groups—*continued.*

Muscle	Ventral	Dorsal
10.—Tensor fasciæ latæ		×
11.—Gluteus minimus		×
12.—Gluteus medius		×
13.—Piriformis		×
14.—Gluteus maximus		×
15.—Biceps femoris (short head)		×
16.—Biceps femoris (long head)	×	
17.—Semitendinosus	×	
18.—Semimembranosus	×	
19.—Adductor magnus	×	
20.—Obturator externus	×	
21.—Adductor longus	×	
22.—Adductor brevis	×	
23.—Gracilis	×	
24.—Obturator internus	×	
25.—Superior gemellus	×	
26.—Inferior gemellus	×	
27.—Quadratus femoris	×	
28.—Tibialis anterior		×
29.—Extensor hallucis longus		×
30.—Extensor digitorum longus		×
31.—Peroneus tertius		×
32.—Peroneus longus		×
33.—Peroneus brevis		×
34.—Extensor digitorum brevis		×
35.—Gastrocnemius	×	
36.—Soleus	×	
37.—Plantaris	×	
38.—Popliteus	×	
39.—Tibialis posterior	×	
40.—Flexor digitorum longus	×	
41.—Flexor hallucis longus	×	
42.—Quadratus plantæ	×	
43.—Abductor digiti quinti	×	
44.—Flexor digiti quinti brevis	×	
45.—Opponens digiti quinti	×	
46.—Interosseus plantaris 1	×	
47.—Interosseus plantaris 2	×	
48.—Interosseus plantaris 3	×	
49.—Interosseus dorsalis 1	×	
50.—Interosseus dorsalis 2	×	
51.—Interosseus dorsalis 3	×	
52.—Interosseus dorsalis 4	×	
53.—Adductor hallucis	×	
54.—Lumbricalis 1	×	
55.—Lumbricalis 2	×	
56.—Lumbricalis 3	×	
57.—Lumbricalis 4	×	
58.—Abductor hallucis	×	
59.—Flexor digitorum brevis	×	
60.—Flexor hallucis brevis	×	

Table III.—Division of the Muscles of the Axis into Ventral and Dorsal Groups.

Muscle	Ventral	Dorsal
Muscles of the Back.		
Serratus posterior superior		×
Serratus posterior inferior		×
Splenius		×
Sacrospinalis		×
Iliocostalis		×
Longissimus		×
Spinalis dorsi		×
Semispinalis		×
Multifidus		×
Obliquus capitis inferior		×
Obliquus capitis superior		×
Rectus capitis posterior major		×
Rectus capitis posterior minor		×
Rotatores		×
Interspinales		×
Intertransversarii	×	
Trapezius		×
Muscles of the Neck.		
Scalenus anterior	×	
Scalenus medius	×	
Scalenus posterior	×	
Longus capitis	×	
Rectus capitis anterior	×	
Longus colli	×	
Rectus capitis lateralis	×	
Sterno-cleido-mastoid	×	
Muscles of the Thorax.		
Intercostales	×	
Levatores costarum	×	
Subcostales	×	
Transversus thoracis	×	
Muscles of the Abdominal Wall.		
Obliquus externus abdominis	×	
Obliquus internus abdominis	×	
Cremaster	×	
Transversus abdominis	×	
Pyramidalis abdominis	×	
Rectus abdominis	×	
Quadratus lumborum	×	
Muscles of the Perineum.		
Sphincter ani externus	×	
Corrugator cutis ani	×	
Transversus perinei superficialis	×	
Bulbocavernosus	×	
Ischiocavernosus	×	
Sphincter urethræ membranaceæ	×	
Transversus perinei profundus	×	
Muscles of the Pelvis.		
Levator ani	×	
Coccygeus	×	

Table IV.—Division of the Nerves of the Extremities into Dorsal and Ventral Groups.

Origin	Nerves	
Upper Extremity.		
Brachial Plexus.	Dorsal trunks (posterior cord)	Dorsal scapular Long thoracic Suprascapular Subscapular (2) Thoraco-dorsal Axillary Radial
	Ventral trunks (lateral and medial cords)	Nerve to subclavius Anterior thoracic (2) Musculocutaneous Median Ulnar
Lower Extremity.		
Lumbo-sacral Plexus.	Dorsal trunks	Superior gluteal Inferior gluteal Nerve to piriformis Femoral Peroneal
	Ventral trunks	Obturator Nerve to obturator internus and superior gemellus Nerve to quadratus femoris and inferior gemellus Tibial

This division of the nerves and, as naturally would follow, of the muscles of the body, is also to be found in part in Braus,[4] Quain's *Anatomy*,[5] and Lewandowsky.[6] Indeed, the whole general notion of such a division was recognized to a certain extent by Thomas Huxley[7] in 1871, in his description of the surfaces of the limbs and their axial borders. Since the functions of these muscles, particularly those of the limbs, are familiar to all, they have not been included in the tables. The difference between an expected function in terms of the dorsal or ventral origin of a given muscle and its real function has been emphasized briefly by citing the femoral group and the dorsal interossei. When this same critical examination of the correspondence between function and origin of muscles is applied to the hamstrings, for example, it is found that whereas they flex at the knee, they also extend at the hip—a curious combination. When the movements at the ankle are considered, it is found that the gastrocnemius and soleus group, though posteriorly placed in the leg, and though developing from what is really its ventral aspect,

have a dorsal function, extension. They are innervated by a ventral nerve which also innervates the flexors of the toes and the intrinsic flexors of the foot. The confusion which has resulted from this alone is tremendous. The attempts to reconcile function on one hand, with anatomy, embryology, and nerve-supply on the other, have led to insurmountable difficulties so far as any satisfactory solution is concerned. However, when in addition to actual movements of muscles, we consider their embryological grouping and their nerve supply, we find that all of these difficulties disappear.

In order to keep in mind this division of muscles, it is proposed that the words 'ventrad' and 'dorsad' be added when necessary to our present terminology to indicate the control and origin of muscle movements such as extension, flexion, abduction, etc.; the suffix 'ad' instead of 'al' is thus used to indicate that primitively these muscles produced movements to and from the ventral surfaces of the body. To be sure, all movements are not primarily such, but a group of movements such as flexion, internal rotation, adduction, and inversion obviously have the same general effect, while the movements of extension, abduction, external rotation, and eversion have the opposite effect.

In another paper[8] emphasis is laid upon the fact that the movements of the leg in normal gait can be resolved into four phases: (1) A ventral fin-like movement at the hip; (2) A dorsal fin-like movement at the hip; (3) The flexion reflex element; (4) The support element.

The first two of these represent movements of the extremities of a vertebrate with but singly-hinged appendages, namely fins. When, in the course of vertebrate evolution, fish developed into land animals, amphibia, and acquired three-hinged appendages, more complex movements were necessary. The three-hinged appendage had not only to be locked in extension to counteract gravity, but had also to be withdrawn from the ground. Land animals therefore developed a more complicated integration of the nervous system to control this. These two latter sets of movements, which are corresponding opposites, include the two last elements of gait, those of flexion and support, and it is these which are made manifest in an uncontrolled fashion by the process of decerebration, either experimentally in animals or by disease in man.

The position of antigravity posture as defined by Sherrington[9, 10] is spoken of as 'extension', while the flexion reflex element is spoken of as 'flexion'. The impression given, therefore, is that these two sets of movements are physiologically homogeneous, and the implication is that the nervous system bears a similar relation in controlling them. However, in the following it will be shown that both the

extension and flexion alluded to consist of an alternation of ventral and dorsal movements, not a simple and homogeneous reaction. These reflexes of posture and progression are, of course, guided by integrations of the spinal cord. Their make-up is obviously more complex than one which would only guide the movements of a large ventral or dorsal group of muscles in a concerted fashion. The postural reaction of the trunk of the body in opisthotonus illustrates by way of contrast a simple over-activity of the dorsal group in relation to the ventral group.

III.—A NEUROMUSCULAR ANALYSIS OF THE EXPERIMENTAL AND CLINICAL EVIDENCE FOR DECEREBRATE RIGIDITY AND FLEXION REFLEX.

A. **The Experimental Evidence.**—In a paper entitled "The Reflex Mechanism of the Step", Sherrington[9] has given the actual

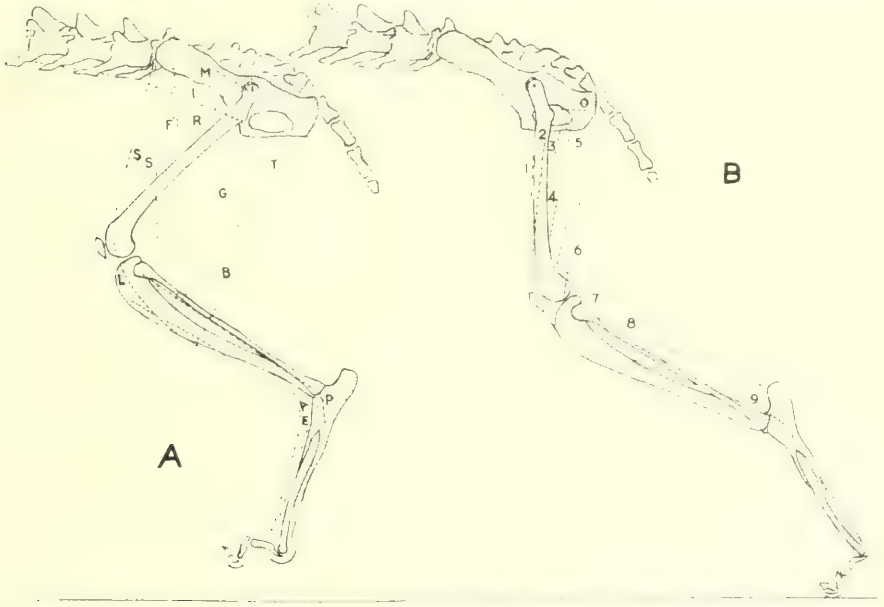

FIG. 1.—Figure illustrating the muscles actually observed by experimental analysis to be engaged in contracting in the flexion-phase (A) and in the extension-phase (B) of the reflex step of the cat.

A

A. Tibialis anticus.
B. Biceps femoris posterior.
E. Extensor brevis digitorum.
F. Tensor fasciæ femoris brevis.
G. Gracilis.
I. Psoas.
L. Extensor longus digitorum.
M. Gluteus minimus.
P. Peroneus longus.
R. Rectus femoris.
S. Sartorius lateralis.
S'. Sartorius medialis.
T. Semitendinosus.

B

0. Quadratus femoris.
1. Crureus.
2. Vasti.
3. Adductor minor.
4. Adductor major (a part).
5. Semimembranosus.
6. Biceps femoris posterior.
7. Gastrocnemius.
8. Solens.
9. Flexor longus digitorum.

By permission of 'Brain'.

Table V.—The Above Muscles Arranged in Ventral and Dorsal Groups.

Note.—In both tables *Group I* includes muscles acting at the hip, *Group II* those acting at the knee, *Group III* those acting at the ankle. *Fl.* Flexion: *Ext.* Extension: *Ad.* Adduction.

	A		B	
Group	Ventral	Dorsal	Ventral	Dorsal
I.		I. Psoas M. Gluteus minimus R. Rectus femoris F. Tensor fasciæ femoris brevis S. Sartorius lateralis S'. Sartorius medialis	0. Quadratus femoris 3. Adductor minor 4. Adductor major (a part) 5. Semimembranosus 6. Biceps femoris posterior	
II	B. Biceps femoris posterior G. Gracilis T. Semitendinosus			1. Crureus 2. Vasti
III.		A. Tibialis anticus L. Extensor longus digitorum P. Peroneus longus E. Extensor digitorum brevis	7. Gastrocnemius 8. Soleus 9. Flexor longus digitorum	

The Corresponding Muscles in Man.*

I	Pectineus (Fl. & Ad. hip)	Iliopsoas (Fl. hip) Tensor fasciæ femoris (Fl. hip) Sartorius (Fl. hip) Pectineus (Fl. hip)	Adductors (Ad. hip) Semimembranosus (Ext. hip) Semitendinosus (Ext. hip) Biceps femoris (Ext. hip)	
II	Gracilis (Fl. & Ad. knee) Semitendinosus (Fl. knee) Semimembranosus (Fl. knee) Biceps femoris (Fl. knee)			Quadriceps femoris (Ext. knee)
III		Tibialis anticus (Fl. ankle) Extensor digitorum longus (Fl. ankle) Extensor longus hallucis (Fl. ankle)	Gastrocnemius (Ext. ankle) Soleus (Ext. ankle) Tibialis posticus (Ext. ankle)	

*This does not account for all the muscles of the lower extremities, since observation or testing of all of them has not been possible.

muscles involved in the extension or antigravity phase of reflex stepping and in the flexion phase in the cat. *Fig.* 1 represents the results[9]; following this is a table (*Table V*) showing the division of muscles into dorsal and ventral groups, and below this, for convenience and future use, the corresponding muscles in man.

From this it is readily seen that there is a definite alternation of control at the three great joints, the hip, knee, and ankle.

Furthermore, in speaking of the muscles of the axis, Sherrington[9] states that "when all of the brain in front of a transection between

anterior and posterior colliculi is removed in a cat or dog, the decerebrate preparation thus obtained exhibits a systemic postural reflex with the following features . . . the dorsal muscles of the back, neck, and tail, the retractors of the head, and the elevators of the lower jaw, are all in harmoniously co-ordinated steady tonic contraction."

From this it may be seen that the muscles of the extremities show an alternation from axis towards periphery at the knee and hip respectively, and, in addition, this alternation is carried on in the axial muscles in which the dorsal group is over-active. The formula, therefore, for the limbs in decerebrate rigidity, going distally, is VDV, and the formula for the limbs and axial musculature, the axial being most proximal, is DVDV. These letters refer respectively to axis, hip, knee, and ankle. This experimental evidence is clearly cut, and forms the basis of a conception of muscular pattern in decerebrate states, to be illustrated later by case reports. Experimentally this pattern is more clearly defined than would be expected in the less uniform effects of human pathological processes.

B. The Clinical Evidence for Decerebrate Rigidity. The first clinical description of decerebrate rigidity was given by S. A. K. Wilson[11] in November, 1920, a quarter of a century after the first experimental description by Sherrington. Wilson showed that a great variety of disorders, such as cerebral abscesses and tumours, cerebellar tumours, intraventricular hæmorrhage, acute purulent meningitis and ependymitis, tuberculous meningitis, hydrocephalus, congenital diplegias, chorea, athetosis, and dystonia lenticularis, could cause a picture resembling this or fragments thereof. A few quotations from his paper will suffice to give the movements affected.

Case 1 (p. 223).—" The arms were extended by the sides, slightly flexed at the wrists and notably rigid. The legs were also extended and adducted, with toes pointed down and in."

" 11.45 p.m. About this time both arms became rigid, the left rather more than the right. Both were adducted at the shoulder and strongly extended at the elbow, with pronounced internal rotation and hyperpronation ; the wrists were flexed, so that the palms of the hands looked up and out, quite turned away from the body." (*Fig.* 2.)

" 1.25 a.m.—When the patient was seen again, after about an hour's interval, he was in the same extensor position, arms adducted at shoulders, extended at elbow, flexed at wrist, whole arm rotated inwards and hyperpronated ; on the right the thumb was between the first and second fingers of the hand, but not on the left. The legs were rigidly extended, feet inverted, great toes dorsiflexed."

Case 2 (p. 227).—" The whole of the musculature of trunk, arms and legs became rigid ; the arms and legs were extended to the fullest extent, the arms by the sides and pronated, while the hands were clenched and wrists slightly flexed. The legs were slightly inverted, the feet more notably so, with the toes pointing down and in."

Case 3 (p. 228). "The upper extremities were adducted and internally rotated, the forearms strongly hyperpronated, so that the backs of the hands faced each other; trunk and neck were extremely rigid and the latter slightly retracted; the legs were similarly in full extension, heels drawn up, toes pointing down and feet inverted."

Case 4 (pp. 229 and 231).—"A brief period of excitement and moaning ushered in the attacks, which consisted of sudden powerful opisthotonus and head retraction, the neck straightening and the occiput nearly touching the shoulders. The left arm relinquished its flexed attitude and became strongly extended, the forearm over-pronated, the wrist extended and the third and fourth fingers flexed, while the thumb was flexed into the palm. The legs were in fullest extension with heels up and toes down, and the right arm was as the left, except that it was rather less inverted and the hand was in the form of a fist. Trunk and limbs alike were absolutely rigid."

Case 5 (p. 231).—"March 6, 1906: The legs were now extended again, and so were the arms. The patient lay unconscious, with the arms extended by the sides and the forearms notably hyperpronated. The hands were clenched. The legs were also fully extended, with feet inverted and toes pointing down and in."

FIG. 2.—The typical decerebrate attitude of extension pronation.
(By kind permission of 'Brain').

Case 6 (p. 233).—"The arms were fully extended, adducted, strongly hyperpronated; the backs of the hands faced each other, and at the same time the wrist and finger flexors were conspicuously contracted; the neck was stiff from muscular rigidity, but was not retracted in any degree; the lower extremities were in full extension and adduction, with heels drawn up, toes pointing down, and feet slightly inverted."

Case 7 (p. 235).—"His arms went out in front of him, extended and hyperpronated, with hands clenched and wrists flexed."

When these descriptions are compared with the tables of movement given by Sherrington and with *Tables I*, *II*, *III*, *IV*, and *V*, it will be seen that the formula D—axis, V—shoulder and hip, D—elbow and knee, and V—wrist and ankle and below, is clearly present.

The quotations from Wilson's paper were chosen to illustrate clearly-defined patterns. Many of his descriptions, as would be expected from the character of the pathological process, were not so

precise. The lack of clearly defined pattern in many cases appears to us susceptible of easy interpretation, as indeed has been suggested by Wilson. In the cases which follow we have fortunately been able to find a precise formula in all but one. In the light of the neuromuscular approach, these formulæ become significantly indicative of curious and inexplicable patterns in other conditions.

C. The Clinical Evidence for the Flexion Reflex.—When the illustrations from Sherrington's article on "The Reflex Mechanism of the Step"[9] are examined, it is seen that the flexor phase, or the flexion reflex element, forms a corresponding opposite to the antigravity position. "A complete flexion reflex consists of a single flexion at the hip and knee, with dorsiflexion of foot and toes."[12]

Reference to the tables added to these illustrations shows that the formula is D—hip, V—knee, D—ankle for the limbs, going distally, in contrast to the VDV of antigravity posture. Furthermore, it has been shown by Walshe,[12] Riddoch,[13] and others that a well-marked contraction of the abdominal musculature occurs with the fully-developed flexion reflex. Therefore the formula becomes VDVD for the musculature of the limbs and trunk, and is in contrast to the DVDV formula of antigravity posture. These letters refer respectively to the axis, hip, knee, and ankle. *Case* 5 described below (*Fig.* 9) illustrates this.

D. Summary.—From this it may be seen that, not only in decerebrate posture, but also in the corresponding opposite, the flexion reflex, an alternating formula exists in the extremities, which is not unexpected, after all, when one considers the optimum mechanical needs of appendages consisting essentially of three segments which must be folded up and extended. This alternation is also present when the axial musculature is included.

IV.—CASE REPORTS.

In the case reports given below, not only movements as such, but the rôle of individual muscles in causing these movements, have been repeatedly and most carefully observed. The axial movements, and all movements of the extremities, except those at the shoulder, are easy to analyze by observation and various clinical tests. Certain difficulties appear at the shoulder, due to the fact that both dorsal and ventral muscles bring about the same movement, notably internal rotation. These muscles are the pectorals of the ventral group, and the latissimus dorsi, subscapularis, and teres major of the dorsal group. However, it is very easy to palpate both the pectorals and the latissimus, and thus determine which of these large muscles is in action.

Case 1. R. E. (*Fig.* 3).

History. In October, 1919, at the age of 15, the patient was taken with a respiratory disorder manifesting itself as a cold and bronchitis, unassociated with expectoration. She complained of pain in the chest. She was treated at various hospitals and dispensaries. The respiratory symptoms cleared up, but the pain remained for almost a year, until Sept. 25, 1920. At that time she had been suffering from excessive drowsiness for a month. She complained of diplopia, headache, fever, drooping of the right eyelid, and dragging of the left foot. At Christmas, 1920, choreiform movements in the legs began which prevented her from sitting still. Two days later similar movements appeared in both hands, and, two days after that, in the neck and head. By this time the diplopia had disappeared, though ptosis and dragging of the left foot still persisted. On Jan. 21, 1920, she was admitted to Bellevue Hospital, where she remained for five months. Three months after admission the ptosis disappeared. While at Bellevue the movements were those seen in the kinetic form of decerebrate posture. A precise analysis of the muscles involved was not made at that time. It was noted then that the functional element was large. The underlying process, however, was felt to be epidemic encephalitis. After discharge from Bellevue and a brief stay at the Metropolitan Hospital, she was admitted to Montefiore Hospital, Sept. 29, 1921.

FIG. 3.—Note the adduction at the shoulders, the decerebrate position of the left arm, and the adduction at the hips.

Physical Examination.—At the present time examination shows bilateral ptosis, nystagmoid oscillation in the lateral plane, and weakness of the lower facial muscles on the right. Standing unassisted is impossible. There is no other involvement of the muscles supplied by the cranial nerves except the spinal accessory, which will be described under axial musculature.

Axis.—There is definite arching of the body with retraction of the head, simulating typical opisthotonus. She has shrugging movements of the shoulder which are produced by the action of the trapezius. The sterno-cleido-mastoid on the right contracts occasionally.

Upper Extremities.—The arms are adducted and rotated inwardly, and the pectorals can be felt to contract when this movement occurs. There is no contraction of the latissimus, the deltoids, rhomboids, or spinati muscles. The forearm is extended on the arm by the triceps, and the wrist is pronated and flexed. While at Bellevue it was noted that the fingers were consistently flexed and adducted during the movements, while here the first finger is extended. The present finger movements imitate classical athetosis.

Lower Extremities.—In the lower extremities there is a constant movement of what appears, on first analysis, to be flexion, and what really is a spasmodic adduction, of the thighs due to definite contraction of the adductors. Occasional contraction of the pectineus is felt (supplied by both

dorsal and ventral nerves). When the legs are abducted there are active adductor movements which bring the legs to the mid-line. During this movement flexion at the hip does not occur. The iliopsoas, sartorius, tensor fasciæ latæ, and glutei do not contract. When the patient is on her abdomen and the knee is flexed at right angles, rhythmic extension movements produced by the quadriceps occur. When she is on her back the hamstrings can be felt to contract. This does not produce flexion at the knee, but rather slight extension at the hip. Rhythmic movements accentuate a rather mild equinovarus position. The toes are flexed in this movement.

Summary. Of the muscles which could be tested, the following are found active and producing movements :—

1. Axial
   - Extensors of the head, neck, and back
   - Trapezius
   - Right sterno-cleido-mastoid
2. Appendicular
   - *a. Upper extremity*
     - 1. Shoulder — Pectorals
     - 2. Elbow — Triceps
     - 3. Wrist — Flexor group; Pronators
     - 4. Fingers — Long flexors; Extensor indicis proprius; Adductor and flexors of the thumb; Volar interossei and lumbricales
   - *b. Lower extremity*
     - 1. Hip — Pectineus; Hamstrings; Adductors
     - 2. Knee — Quadriceps
     - 3. Ankle — Gastrocnemius; Soleus; Tibialis posterior
     - 4. Foot — Long and short flexors; Abductors

With the exception of the sterno-cleido-mastoid, only dorsal axial muscles come into play. Movements at the shoulder and hip are brought about by ventral muscles, those at the elbow and knee by dorsal muscles, those at the wrist and ankle by ventral muscles. In the fingers and toes the movements are ventral except that of the extensor proprius.

Diagnosis.—Epidemic encephalitis, choreic and kinetic type.

Case 2.—L. R., age 13 (*Figs.* 4 and 5).

History.—The patient was admitted to the Montefiore Hospital on April 8, 1922. The history obtained from the mother was that the child had not been well for about two years. She was irritable as a rule, easily disturbed, and fainted frequently. The mother believes she may have been feverish at times. There is no history of diplopia (?) In February, 1922, she was brought home because movements of the limbs, head, and trunk had begun. After staying a month in another hospital she was admitted to Montefiore Hospital.

Physical Examination.—On first sight she presents the picture of chorea. The arms, legs, and head are moving almost continually. The

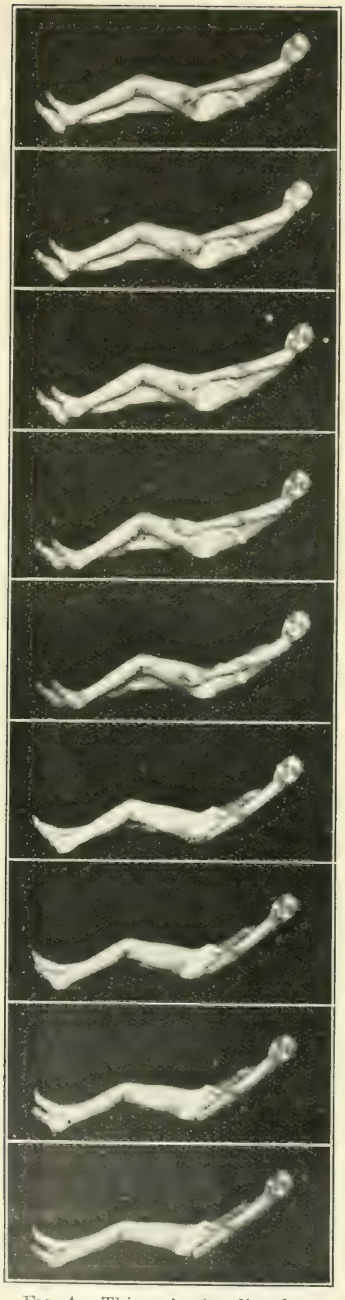

Fig. 4.—This series (reading downward) shows the arm passing from a decerebrate position (adduction of the shoulder, extension at the elbow, hyperpronation at the wrist) to one of rest. The left leg passes from adduction to rest at the same time.

duration of the movement is less than a second when timed by a stop-watch. More careful observation shows that certain definite groups of muscles are brought into activity, thus producing a continual repetition of the same postural patterns. When the patient is asleep there are no abnormal positions or postures. With the exception of the movements noted, the physical examination is quite negative. The cranial muscles are negative except for those supplied by the spinal accessory, which will be mentioned below.

Axis.—The extensors and rotators of the head bring about retraction and rotation. Slight opisthotonic movements are occasionally noted. When the patient is more excited, these movements become more pronounced. The trapezius and sterno-cleido-mastoid are occasionally contracted on both sides. With the exception of this latter muscle, no activity of the ventral muscles of the axis has been noted.

Upper Extremities.—When the patient is relatively quiet, the pectoral group at the shoulder adducts and internally rotates the entire arm. As the series of moving pictures shows (*Fig.* 4), this movement alternates with one of rest. At the elbow the triceps extends the arms. At the wrist the flexors are in action as well as the pronators. The flexors of the fingers and thumb, as the photograph (*Fig.* 5) and moving picture show, are also in action.

It may be seen from this that there is an alternation of activity of ventral and dorsal muscles, the formula of which is at the shoulder ventral, at the elbow dorsal, at the wrist and below ventral.

Lower Extremities.—The legs are carried forward and across the mid-line by the ventral adductor group. The iliopsoas is not in action, as can be plainly made out on examination. When the legs are widely spread, flexion at the hip is not seen; instead, the legs are adducted. Extension at the hip by the hamstrings is also present. The gluteal group of muscles is not in action except when the axial muscles are bringing about opisthotonus. At the knee the hamstrings produce no flexion; on the contrary, the dorsal quadriceps group extends. At the ankle the gastrocnemius and soleus and tibialis posticus produce extension, and the

flexors of the toes are active. As reference to *Table III* will show, this last is entirely a ventral action.

The formula for the leg is therefore identical with that of the arm: at the hip ventral, at the knee dorsal, at the ankle and below ventral. When the patient's movements become excessive owing to excitement, the pattern ceases to be so precise, and other muscles begin to contract. These muscles are the antagonists of those noted above. This is a relatively rare occurrence.

Summary.—The patient shows activity of the dorsal musculature of the trunk, and an alternating formula for the extremities. The only exception is the sterno-cleido-mastoid. Occasionally the antagonists of these muscles go into action.

Diagnosis.—Epidemic encephalitis, choreic and kinetic type.

FIG. 5.—Note the right arm and the adduction of the right leg. Compare with *Fig.* 2.

Case 3.—J. S., age 17 (*Figs.* 6 and 7).

History.—This patient was admitted to the Montefiore Hospital on May 1, 1922. She has had a rather stormy career, but despite family opposition had worked hard in pursuing her studies at high school. She graduated in January, 1920. She then studied at a business school, graduating at the head of her class. She began stenography, and since has done exceptionally well.

Between January and July, 1920, she began to feel tired and sleepy during the day, and restless at night. She could not sleep well. In July, 1920, she went to the country, but did not improve, and so returned home after two weeks. In August, 1920, she had pains in her legs, with fever, lasting one week. In January, 1921, there occurred a family quarrel centred about a sexual affair, and followed by a disturbed night. In the morning she noticed movements of the hands, feet, and head. She could not talk. Some improvement occurred, but two days later diplopia and fever were present. She soon became delirious, and was admitted to the Lenox Hill Hospital, where she stayed three months. After a stay in the country her general condition improved. In May, 1921, after an attack of vomiting associated with abdominal pain, she was admitted to Bellevue. Following this her movements were intensified, and she was admitted to the Neurological Institute, where she remained nine weeks. There was considerable improvement, but she continued to attend the dispensary until February, 1922. In March, 1922, she entered Mt. Sinai with abdominal pain and depression. She became very noisy and excited, and was occasionally confused, especially at night. She finally left the hospital and was admitted to Montefiore Hospital May 13, 1922.

Physical Examination.—On first observation the patient's condition suggests chorea. The extremities and head are moving continually. These movements are not so pronounced when she is calm and unobserved. The emotional reaction resultant upon examination intensifies the movements. Careful observation shows that the activity of certain groups of muscles predominates and produces a repetition of the same postural pattern.

Cranial Nerves.—When the patient is quiet, the motor cranial nerves are also at rest. When excited, there is frequent grimacing, the tongue is protruded straight forward and to the right and left.

Axis (*Fig.* 6).—There are opisthotonic movements. The head is pulled backward, the spine is curved. The patient states that sometimes

Fig. 6.—Note the opsthotonic movement in particular.

Fig. 7. Note the positions of the arms, hands, and feet.

this begins at the neck and passes wave-like to the end of the body. At other times either the neck region or the lumbar region is alone affected. The trapezius is found active on both sides, but more so on the right. It elevates the shoulder at times. The sterno-cleido-mastoid on the right is sometimes in action, though rarely. The lower ventral trunk muscles (abdominals) occasionally contract, but never bring the body forward.

Upper Extremities.—Some of the shoulder muscles are active. The scapula is moved out and up. On analysis this is found due to the trapezius and to internal rotation by the pectorals. The deltoids and rhomboids are never in action. Occasionally a contraction of the latissimus dorsi is observed. The triceps is almost always contracted, the elbow flexors are rarely so. Flexion occurs at the wrist, though extension is also observed at times. Pronation is frequently noted, but supination also

occurs, though less often. When extension is present supination is noted also. The fingers do not show as typical or as constant a response as that which occurs in *Cases* 1 and 2.

Lower Extremities. The dorsal muscles at the hip do not take part in any of the movements. The patient states that her leg is never carried forward, either when standing or in bed. None of the following muscles contract in any of the movements: iliacus, psoas major and minor, pectineus, sartorius, and tensor fasciæ latæ. The glutei do not contract while standing or when on the abdomen or back. The adductors are powerfully active, and may be felt when prone, supine, or standing. This movement appears one of flexion, but is not in the dorsoventral plane but at an angle tilted towards the mid-line. When the patient is on her abdomen, extension at the hip is produced by the hamstrings, while the glutei are definitely relaxed. The patient states that her leg is twisted inwards (inwardly rotated). She does not confuse this with inversion of the foot. The leg is continually held in extension, and never when standing or prone goes into flexion. The feet are inverted, the right one more so than the left, and extended so that the dorsum of the foot is almost in a straight line with the long axis of the tibia. When standing, both feet are inverted, the right much more than the left. When walking, the external lateral border of the foot is turned to the ground owing to inversion (*Fig.* 7). This can be momentarily overcome, but immediately returns.

Summary.—In this case of epidemic encephalitis there is a well-defined picture showing a rhythmic recurrence of opisthotonus and the antigravity posture. When the patient is quiet this is easily made out. When excited there is overflow into the opposite pattern, though even then it occurs much less frequently than that of the antigravity posture.*

Diagnosis.—Epidemic encephalitis, choreic and kinetic type.

Comments on Cases 1, 2, and 3.—The diagnosis in the three cases is epidemic encephalitis of the kinetic and choreic types. It is extremely interesting that at one time or another each of these three cases was diagnosed as hysteria. The hysterical element, or what might better be considered functional overflow, is marked. Were it not for the findings in the case of R. E., namely, paresis of one leg, persistent and partial ptosis of both eyelids, diplopia, nystagmus, headache, and fever; for the prolonged illness in L. R., with fever and delirium; and the insomnia, fever, diplopia, and intestinal and bladder disorders in J. S., these three patients could be considered as hysterics. However, it is now well known that the lesions of epidemic encephalitis not only produce pictures which are subject to great exaggeration by mental factors, but that the disease may be practically latent until mental shocks occur. There is no difference, in the effect of physical and psychic trauma in bringing this disease to light, from what familiarly occurs in other diseases of the nervous system, such as paralysis agitans and tabes dorsalis.

* At the end of August the contractures at the ankles and of the toes had so increased and the movements had become so marked and chaotic as to give the picture of dystonia lenticularis.

The Muscular Element. In all three cases the muscles involved in producing the patterns described were almost identical. Indeed, if it were not for a few exceptions, the descriptions of any one of the three cases would fit the others. In all of them the dorsal axial muscles were instrumental in causing movements. In all of them the muscles described by Sherrington, and then by Wilson, as producing patterns of decerebrate posture, were in action. It is extremely interesting that those muscles which are engaged normally in maintaining an erect posture should appear as clearly as they did. The formula for the arms and legs was, in all three cases, ventral movements at the hip and shoulder, dorsal movements at the knee and elbow, and ventral at the wrist, ankle, and below. In addition, the ventral axial sterno-cleido-mastoid muscle was active in all three cases. We are unable to explain this to our satisfaction, but feel that it may have to do with separate control by the nervous system of cephalo-rotatory movements. Further study of this matter will have to be made.

The alternating formula VDV of the extremities was complicated in *Cases* 2 and 3 by the appearance of a reverse formula, DVD. Occasional abduction of the arm and flexion of the elbows was seen (*Cases* 2 and 3). More frequent than this were movements of extension and supination at the wrist (*Case* 3). These movements were carried out by dorsally innervated muscles. Further discussion of this flexion reflex formula in the arm appears in Section V.

The movements of the fingers in L. R. and in R. E. in the earlier periods of their illness were definitely flexor. Later on, some of the finger movements involved extension at the metacarpophalangeal joints. In J. S., movements of extension and flexion were somewhat mixed. That this was not unexpected will be later referred to in Section V, where the finger movements in both the antigravity and flexion reflex arm postures receive more attention.

Case 4.—Herbert S., age 28 (*Fig.* 8).

History.—The patient, a cutter, was admitted to Montefiore in April, 1921, and died in October, 1921. He was admitted with the following history. Following a 'cold' in October, 1920, which was associated with gastro-intestinal symptoms, there occurred thermo-hypæsthesia in the lower extremities. Both legs below the knees felt cold when taking a warm bath. A week later stiffness of the left leg occurred, and the next morning dribbling of urine. He then entered the Neurological Institute, and after three weeks noticed some improvement. Soon after, fever developed 101° to 104° associated with swelling of the left elbow. This lasted three weeks. Following this he was unable to walk or stand, though he could move his feet in bed. At this time the sphincters were more severely involved, with bladder incontinence and retention of fæces. Two weeks prior to admission he again had a temperature for two days, and could not move either his arms or his head. This was associated with pain in his left shoulder and side.

Physical Examination. The patient is confined to bed and unable to move unaided. The motor cranial nerves are normal. There was, however, in a previous examination, slight right central facial palsy. The head is rigidly maintained in a normal position when lying down, but when sitting up he can both hold it up and move it in all directions. He is unable to maintain a sitting position by himself, owing to paralysis of the axial musculature.

Upper Extremities (*Fig.* 8). Voluntary movements in the upper extremities are lost, with the exception of movements which carry the arms from a horizontal position upwards from the bed. Flexion and extension of the fingers of the right hand, and flexion of the fingers in the left hand are also absent. The arms are rigidly extended. The pectoral muscles pull the arms forward to the chest. They are very hypertonic. The arms are held extended on the elbows by the triceps on both sides. On the right the biceps shows normal tone, while on the left it is extremely hypertonic. This does not produce flexion. On the right the forearm is pronated so that the ulnar border of the hand faces directly upward. On the left this pronation is only carried to an angle of 45° to the horizontal plane. On the right the wrist is flexed to a right angle. The fingers are forcibly extended at the two terminal phalangeal joints. When flexed by the examiner they returned to the extended position with an elastic-like bound. The thumb is adducted and slightly flexed at the proximal joint. The fingers are adducted. The left hand is *extended* at the wrist, nearly at right angles, while the fingers are flexed at the first interphalangeal joint. The gradation of flexion passes from about 5° at the index finger to 90° at the little finger. The thumb is flexed at the proximal joint and adducted into the palm. The position of the left hand is much like that due to the associated movements resultant upon making a fist, that is, flexion at the fingers and extension at the wrist.

FIG. 8.

Lower Extremities.—These are both rotated so that the lateral plantar borders of the foot rest upon the bed. They are flaccid, and present only the defects due to loss of tone and voluntary power, notably flexion of the toes and of the sole of the foot, with extension at the ankle (pes cavo-equinus).

Sensation.—Lost from the third dorsal segment downward.

Reflexes. The jaw-jerk is extremely lively. The deep reflexes are not obtained in the arm. The abdominal reflexes are absent. The knee-jerks are diminished.

Throughout the examination the patient showed 'flexor spasms' of the legs. The slightest stimulus caused this retraction. It has been necessary to hold the patient's legs down by a sheet to prevent these seizures. Once during the examination, reflex priapism was noted. Control of the bladder is lost.

Spinal Fluid.—This shows the following: increased pressure; globulin +++; flocculent precipitate; reduction to Fehling's diminished; cells 70.

Wassermann.—Blood and spinal fluid negative; blood culture and spinal fluid negative.

Urine.—Negative.

Summary.—The patient presented a varying course, and finally died a bulbar death. He ran a septic temperature throughout his stay in the hospital. A diagnosis of severe infective meningo-encephalomyelitis was made. The flaccidity of the legs and the sensory changes from the third dorsal segment downward, coupled with the reflex spasm, reflex priapism, and reflex incontinence, indicated a very severe lesion in the upper dorsal cord. The extremely spastic paralysis of the arm muscles, the transient facial palsy, and rigidity of the neck indicate that the process involved higher portions of the neuraxis.

Comments.—We have used this case to illustrate a static hypertonic condition in the upper extremities, showing difference in posture on the two sides. Though the condition of the lower extremities is pertinent to the subject of this paper, we shall leave a discussion of this matter for the next case. The points which we wish to emphasize are that: (1) The state of the upper extremities was unchanged for a considerable length of time. (2) No choreic movements were present. (3) The asymmetry in the two hands, as showing in *Fig.* 8, was very striking. It indicated that the process must have involved slightly different centres as far as the forearm and finger muscles were concerned. On the side in which pronation was most marked, flexion of the wrist was complete. On the side where pronation was relatively slight, extension at the wrist was present. (*See also* Section VII.)

Case 5.—Harry S., age 36, metal worker (*Fig.* 9).

History.—The patient was admitted Sept. 29, 1921, to Montefiore Hospital, complaining of weakness in both limbs, constipation, and difficulty in passing urine. In August, 1919, he complained of 'stomach trouble', which consisted of pain in the 'stomach' and a feeling of a constricting band around the abdomen. In May, 1921, he noticed pain in the knees, which in a few weeks crept upward to his hips. It was more intense on the left than on the right. This pain remained, and in July he experienced what he describes as a 'freezing sensation and pins and needles' in the soles of his feet. Simultaneously weakness was noticed. A lumbar puncture was done, after which he lost the power of using his legs completely. Urgency of urination appeared, as well as constipation, more marked than before. In the early part of August his legs began to stiffen in extension, and grew progressively worse.

Physical Examination. On admission, the cranial nerves were negative. The deep reflexes in all the extremities were exaggerated. The upper abdominals were present, the left lower easily exhausted and the right not obtainable. There was marked spasticity of the lower extremities. Knee- and Achilles-jerks were hyperactive. There was bilateral ankle-clonus, with a Babinski on the left, questionable on the right. Sensory loss from D7 downward existed. He always lay with the lower extremities in extension until after the operation performed by Charles Elsberg on Nov. 26, 1921. *After the operation* the abdominal muscles contracted, the thigh was flexed on the hip. The muscles taking part in the contraction were the whole femoral group. The foot was at right angles to the leg (*Fig.* 9), the entire picture representing the flexion reflex described by Walshe[12] or the nociceptive reflex of Sherrington,[9] or paraplegia in flexion described by Babinski. Knee- and ankle-jerks were active.

Part of Elsberg's operative notes follow: An extradural tumor was found lying mostly on the the posterior aspect of the dura and extending

FIG. 9.—Note the position described as 'flexion at the three great joints' or 'paraplegia in flexion'. The formula is: dorsal at the hip, ventral at the knee, and dorsal at the ankle. This ankle position was more conspicuous on examination than is shown here.

beneath the 4th, 5th, 6th, 7th, and 8th dorsal vertebræ. A large amount of it was excised, but fragments remained in various places in front of the dural sac. There was no doubt that there was more tumor below the parts exposed.

Diagnosis. Endothelioma (?).

Comments.—This patient showed typical hypertonic paraplegia in extension before operation, and typical hypertonic paraplegia in flexion after it. The increase of tone was as great after the operation as before. *This indicates clearly that tone flows into pattern.* Pattern changed in this case from that of the antigravity reflex to that of the flexion reflex. The tumour being cervical, it must have damaged the control of *lower* integrations. This indicates that the pattern of decerebrate posture of the legs must be carried out by

neurones lying within the spinal cord. The presence in the legs of decerebrate rigidity is almost an exact counterpart of an experimentally-produced picture. Why the corresponding opposite pattern developed after operation is not clear. The case illustrates the successive static fixation of two spinal patterns of opposite muscular formula, the first VDV, the second DVD. (*See also* Section VII.)

V. THE FLEXION REFLEX POSITION IN THE ARM.

Up to now emphasis has been laid upon the formula for the legs in the corresponding opposite positions of antigravity posture and the flexion reflex. This has been examined in the light of the experimental work of Sherrington and the clinical work of Wilson and ourselves. For the arms, the formula for the position corresponding to the antigravity position in the legs has been illustrated in the case reports and discussion. However, the position in the arms which would correspond to the flexion reflex element in the leg has not been defined. This, as would be expected, would present a formula opposite to that of the decerebrate arm—notably, dorsal at the shoulder, ventral at the elbow, and dorsal at the wrist. In the course of our investigation of this matter a lengthy paper by Riddoch and Buzzard,[14] which considered this subject, came to our attention. They describe a flexion reflex of the upper limb as follows: "The reflex response, for which the convenient term is 'flexion reflex of the upper limb', was readily obtained in No. 5. On scratching the palm of the paralyzed hand there occurred *flexion of fingers*,* wrist, and elbow, slight abduction and external rotation of the upper arm, and elevation of the shoulder. These were the main components of the general response, which varied in details according to the situation of the stimulus within the receptive field" (p. 434). However, in another place (p. 421), in describing associated movements following stimulation, the effect upon the upper limbs is given as follows: "The upper limbs became rotated outwards at the shoulders, flexed at the elbows, *extended at the wrists with slight supination of the forearms: the fingers became extended at the metacarpophalangeal joints and flexed at the interphalangeal joints, while the thumbs were extended and abducted*".*

An analysis of this long paper will not be made here. Suffice it to say that, if the formula for the 'extension reflex' or the antigravity posture is VDV, as is indicated clearly by the findings of Wilson, Riddoch and Buzzard, and ourselves, it would be presumed that the corresponding opposite to this would have an opposite

* Italics by the authors.

formula and would conform to the movements described in the quotation last given. It would be expected that there would be abduction at the shoulder, flexion at the elbow, extension at the wrist and at the metacarpophalangeal joints. This brings up a matter of considerable importance in the analysis of reflex patterns at the distal joints of the limbs which bears not alone on the question of the upper extremity, but also upon the Babinski reflex and its associated phenomena in the lower extremities. The Babinski phenomena will be given special consideration in another paper.

The intrinsic muscles of the hand are all of ventral origin. Adduction and abduction of the fingers, extension at the phalangeal joints, abduction and adduction of the thumb and little fingers, are all brought about by these ventral muscles. When, therefore, there occurs activation of a pattern which demands a dorsal reaction in the long muscles acting on the wrist and fingers, and when the impulse activating this pattern flows into the intrinsic muscles of the hand, no dorsal intrinsic muscles are there. In the antigravity position the long flexors of the wrist and fingers *and* the intrinsic muscles of the hand are activated (ventral muscles). In the flexion reflex element the long extensors of the wrist and fingers (dorsal) are activated, while the intrinsic hand muscles (ventral) are not. This last produces extension at the wrist, extension at the metacarpophalangeal joints, abduction of the thumb, and, in some instances, extension of the phalanges. Contraction of the extensor digitorum may produce this, depending upon the strength of the stimulus. Consequently the typical picture may include either extension or flexion at the phalangeal joints. The long flexors of the fingers are not active in producing flexion, the volar interossei and lumbricales are opposed by the extensor digitorum communis, the adductor of the thumb by the long abductor.

The postural pattern of the flexion reflex of the arm consists therefore of dorsal activity at the shoulder, ventral activity at the elbow, and dorsal at the wrist and fingers.

At the shoulder the large number of dorsal muscles makes it possible to have a number of different reactions. The following from Riddoch and Buzzard[11] indicates the appearance of the reaction which we have just defined, and also the possible variations: "The movement most commonly obtained consists of adduction and external rotation at the shoulder, flexion at the elbow, and extension of the hand and fingers, but the response varies in a remarkable manner with alternation in the locality of the stimulus. Thus, when the reaction is excited by scratching the palm of the hand, the movement at the shoulder is abduction and retraction of the upper arm. Stimulation of the back of the forearm yields adduction at the

shoulder, and of the inner aspect abduction at this joint. Again, when the stimulus is applied to the skin over the deltoid, the response is mainly strong elevation of the shoulder with adduction and external rotation of the upper arm" (pp. 437, 438). This emphasizes the variations found.

The pattern which we have described for the flexion reflex of the arm appeared in *Case* 3 (J. S.). It occasionally followed or alternated with the antigravity posture. This represents the alternating stepping movements (reflex walking) produced experimentally by Sherrington. It has also been seen in a man suffering from spastic tetraplegia, shown at autopsy to be due to pressure upon the lower brain-stem of an apricot-sized aneurysm of the right vertebral artery.

In the movements of athetosis, dystonia lenticularis, and chorea, these patterns, often fragmentary, can be made out, as Wilson has emphasized. The positions sometimes change so rapidly that it is only by means of successive cinematographic pictures that the analysis can be made.

VI.—THE RELATION OF POSTURE PATTERNS TO TONE.

In this paper it is assumed, that not only the integration which produces the flexion reflex, but also that producing the corresponding opposite pattern, notably the pattern of antigravity posture or reflex standing, is entirely spinal. In order to defend this assumption it will be necessary to consider some of the experimental work undertaken in producing decerebrate preparations. Sherrington[9] states: "This reflex standing disappears when the transection of the brain is made behind the posterior edge of the pons", and "the experiments of Horsley[15] and Thiele show that decerebrate rigidity is hardly seriously impaired by successive sections of the cerebellar region until the paracerebellar nuclei are invaded". Wilson,[11] in his paper on decerebrate rigidity in man, states in discussing the pathogenesis of this condition: "In the first place there is general agreement that decerebrate rigidity makes its appearance after transection of the neuraxis at the level of the anterior colliculi, and disappears by a second section below the neuronic level of the medulla". Sherrington[9] has stated: "When in the dog the spinal cord is severed in the thoracic region, the hind-limbs cannot at first stand; but after lapse of weeks or months they exhibit this power. That is, with hind-feet on the ground the reflex tonus of limb-extension suffices to bear the weight of the limbs and superincumbent hind-quarters. The attitude thus exhibited indubitably amounts to standing, and is sometimes maintained for minutes at a time".

It would appear from these four quotations that, in the first place, removal of the central nervous system down to a point "behind

the posterior edge of the pons" still permits the existence of reflex standing or antigravity posture, and the fourth quotation above indicates that this same reflex position may be obtained in an animal whose cord has been transected in the thoracic region. It is furthermore true that *no difference results in the pattern obtained when the brain is transected successively from the anterior colliculi to a "point behind the posterior edge of the pons"*. In other words, no change in pattern is produced by such successive removals of brain tissue. When a section is made below the point at the posterior edge of the pons, or, to use Wilson's words, the "neuronic level of the medulla", the rigidity disappears. But this is due to a transitory loss of tone, as has been suggested by both Sherrington and Wilson, caused by the removal of the grey matter in the regions between the pontine section and the spinal section. However, as the above quotation of Sherrington notes, after a lapse of time the antigravity posture pattern reappears, even in decapitated animals.

Since the pattern definitely exists when all portions of the brain stem above the pons are removed, the neurones producing it and released by the removal of higher centres must lie in the intact portions of the nervous system, that is, in the spinal cord and small portions of the medulla. A further proof of this is that section of the cord at the thoracic level allows the same antigravity pattern to remain in the segments below.

Tonic influences which activate muscles in this particular pattern appear to lie both in the medulla and in the afferent fibres of spinal nerves. If either or both of these influences are removed, there ensues a period during which the pattern disappears and flaccidity results. However, the following quotation from Graham Brown[16] indicates that proper stimulation from above may still activate the pattern: "The reactions (of decerebrate attitude) may occur many months after division of the dorsal spinal roots of the arm. That is to say, appropriate stimulation in the region of the mid-brain may evoke an extensor postural tonus or a flexor postural tonus. Sherrington has found that the decerebrate rigidity which occurs after removal of the cerebrum does not occur in a 'de-afferented' limb, but the fact that a condition which at any rate very closely resembles this state may be evoked in such limbs seems to point to the conclusion that the absence of this postural tonus in the decerebrate 'de-afferented' animal is due to the failure of the ascending impulses from the limb which normally play—however, indirectly—upon these mechanisms of the mid-brain, and that the mechanisms themselves, if properly activated, are still able to induce the tonus".

Pattern and tone constitute two different entities in the nervous system. Tone is the common factor of hypertonic states, and activates

muscles in various patterns. The experiments of Graham Brown (decapitation and de-afferenting) indicate this. When tone is removed, the pattern, like a picture on the wall of a dark room, is not seen but is none the less there in spite of the fact that it is not activated. In brief, tone flows into moulds of patterns much as an electric current. Just as a galvanic current activates flaccid muscles, so does tone, but in addition, it activates them in pattern.

From a consideration of the muscles involved in the flexion and extension phases of gait, as shown by Sherrington, and from all the work on reciprocal innervation, it would be assumed that the neurones for the flexion reflex integration and the reflex standing integration, being corresponding opposites, would be located in the same portions of the nervous system.

From the point of view taken in this article, based upon a neuromuscular conception of movements, the same conclusion can be drawn. From an evolutionary point of view a similar hypothesis must be formulated. When, in the course of evolution, an animal with singly-hinged, fin-like appendages developed appendages with three joints showing the corresponding opposites, the reflexes of flexion and of antigravity posture, it would be natural to assume that the neurones regulating these two newly-appearing reflex activities would lie in the same portions of the nervous system.

VII.—THE KINETIC AND STATIC TYPES.

The spinal neurones must be activated, or no pattern would be present at all.

In the kinetic types described, the choreiform movements produced the patterns with great consistency. The muscles of the opposite group contracted occasionally (*Cases* 2 and 3). In some cases the kinetic impulse, flowing into lower centres, may produce an apparently patternless picture, as is frequently seen in chorea. These movements, due to their speed, are patternless on ordinary observation, but can, by means of analysis by successive moving pictures, be resolved into definite patterns. In our cases an integration having a clearly definite pattern was exposed to the kinetic impulse. When the opposite of this pattern was activated, it indicated that corresponding opposites were both exposed to the kinetic impulse. Indeed, in reflex stepping we see such a combination beautifully illustrated. Since these opposites constitute a pair of neurone arrangements of the same phylogenetic age, it is natural to find them closely associated.

In the purely spastic variety, *Case* 4, it was found that the tone overflowed not only into those muscles which determined the pattern (triceps), but also into the opposing muscles (biceps). Nevertheless the pattern remained. It would appear, therefore, that though

this tone was distributed to antagonists, *it was distributed subject to pattern. This serves to emphasize the fact that tone flows into pattern and constitutes a separate entity in the nervous system.*

VIII.—SUMMARY AND CONCLUSIONS.

Abnormal movements and positions are customarily described as such without naming in addition the muscles which produce them. There the matter is allowed to rest for all practical purposes—"the arm and hand are flexed"—"the arms are adducted, the elbows extended, the wrist and fingers flexed". True, certain formulæ for movements exist, such as the flexion of the arm and the extension of the leg of hemiplegic contracture, but the *anatomical relations* of the muscles and nerves causing such groups of movements have never been seriously considered in clinical neurology. Therein lies an error of omission. To draw conclusions about the nervous system from descriptions of movements and positions *alone* is an error of commission.

In this paper it has been shown that there is a well-defined grouping of nerves and muscles under integrating control of the spinal cord, and that it is on the basis of this grouping that formulæ expressing disease in certain parts of the central nervous system of man must be built.

The question of classification of spinal integrations is too lengthy to append here. Suffice it to say that the dorsoventral integrations of the axis (opisthotonic, emprosthotonic) and appendages (rod-like movement of the leg in walking), and the alternating VDV–DVD integrations of the appendages, are those concerned in stepping.[8] In these formulæ the three initials describe respectively the type of movements at the hip and shoulder, knee and elbow, wrist and ankle and below. The alternating formula VDVD–DVDV, which includes both the axis and appendages, is made manifest by decerebration, as we have shown (Section III, *C*, and Case Reports).

To say, in describing the antigravity posture of decerebration, that the leg is *extended* at the hip, knee, and ankle, the foot *adducted*, and the toes *flexed*, gives no notion of plan or order. To say that there is *adduction* at the shoulder (though *extension* at the hip), *extension* at the elbow and knee, *flexion* and *pronation* at the wrist (though *extension* at the ankle), gives no notion of plan or order. When the facts relating to the grouping of nerves and muscles, not of movement alone, are inserted into the analysis, it is found that the leg and arm formulæ are the same—VDV—and there appears a simpler plan.

Similarly, when the corresponding opposites are considered, the so-called 'flexion reflex' of the arms and legs, there results the same confusion from a consideration of movements alone. The lower

limb is *flexed* at the three great joints, the toes are *extended*, the arm is *abducted* (though the hip is *flexed*), the elbow and knee *flexed*, the wrist *extended* (though the ankle is *flexed*). Here again clearness results from using what is really old information, the basis of which may be found in such text-books of anatomy as Quain[5] and Cunningham[3]. The formula for both arm and leg is DVD, and is opposite to that of the antigravity posture.

The integrations of the spinal cord have clearly defined patterns. By virtue of their activity certain muscles always act together and in the same general way. Their activity may be recognized not only by inspection and palpation, but by changes in position.

The activating forces of these patterns are two—one kinetic and the other static. The latter is familiarly spoken of as tone. Either of these forces activates muscles in clearly defined patterns, provided that a group of integrating neurones is completely released. When a particular integration is patchily involved, such pictures as those of *Case* 4 result. In any event, neurones causing pattern and neurones causing tone are not the same.

From the points of view of the physiologist and the pathologist, it is of some importance to recognize that the pattern for the trunk and appendages in decerebration is resident within spinal neurones. It represents the highest spinal integration. The fact that it may appear when lesions are produced as high as the anterior colliculi makes it seem probable that, between that level and the upper level of the spinal cord, neurones producing posture patterns for the trunk and appendages different from those of the spinal cord do not exist. These high lesions simply release the spinal patterns.

Integrations—as, for example, those of the proprioceptive system (labyrinth, cerebellum)—do exist above the upper level of the spinal cord.

Building upon the anatomical grouping of nerves and their end-organs, the muscles, we have gradually come to see an increasing complexity of neurone patterns or arrangements, a manifestation of evolution, of which the dorsoventral integration of progression by hip and shoulder movements (tetrapodal animals) forms one stage, and the folding and unfolding DVD and VDV integrations made manifest in the last two phases of stepping and in decerebration form another and later stage.

With a clearly-defined notion of these and the other simpler spinal integrations established, the study of supraspinal integrations of movement and posture and their significance becomes possible.

We wish to express our thanks to Dr. S. P. Goodhart, Chief of the Neurological Service, for permission to make use of the records of the department.

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PONTO-BULBAR CRISES ASSOCIATED WITH SIALORRHŒA IN SYPHILIS OF THE NERVOUS SYSTEM.*

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I.—INTRODUCTION.

The objects of presenting this case are to emphasize the fact that crises may occur in cases of cerebrospinal syphilis other than tabes dorsalis, and to describe a group of symptoms referable to the 7th, 9th, and 10th cranial nerves.

It is customary to associate crises with tabes. However, there seems no reason to believe that involvement of nerves or nerve centres sufficient to produce crises cannot occur without concomitant signs of tabes. Nonne[1] emphasizes this in discussing gastric crises. He says, "It is well known and has recently been re-emphasized by Dunger, that gastric crises may be the initial symptoms of tabes. There is no question as to the diagnosis of tabes when pupillary disturbances, absence of Achilles or of knee-jerks or of both, occur in such cases. This is not so in those cases in which 'characteristic gastric crises' occur at short or long intervals in those who have been syphilitic without any objective organic sign as far as the nervous system is concerned. I know of three such cases, and have been able to follow the first case for four years, the second and third cases for three years. All three patients had been syphilitic. Examination of the spinal fluid in all three showed a mild grade of lymphocytosis. I had not begun testing for globulin when I was first consulted by these patients. As you will hear presently, a mild grade of lymphocytosis in the luetic is not sufficient for the diagnosis of tabes."

The case here reported presents a rare type of mixed sensory, secretory, and motor crisis which is scarcely mentioned in the current text-books, and of which the most conspicuous sign—profuse paroxysmal salivation—is only sparingly discussed in the literature of crises.

* From the Medical and Neurological Divisions of Montefiore Hospital, New York. Read before the Section of Neurology and Psychiatry of the New York Academy of Medicine, April 11, 1922.

II.—LITERATURE.

The first note of salivation in tabes which we have been able to discover is in Pierret's[2] thesis of 1876. Six years later Putnam[3] reported six cases of tabes with salivation. The salivation was usually associated with some other form of crisis. One of the patients had occasional attacks during which the saliva "flowed like a fountain" for about fifteen minutes. In the case of another patient each attack of salivation lasted about seven hours. Then Pitres[4] described a tabetic who had had gastric crises which disappeared after a few years. Some time afterward an intense salivation developed which resisted all treatment. It appeared suddenly in the morning soon after the patient arose, and lasted two to three hours; it was accompanied by a sense of heat in the upper part of the face and in the tongue. For a period of two weeks the salivation was continuous day and night. It finally ceased, but recurred two years later. There was no disturbance of taste, but the sense of smell was completely lost. In 1887 Féré[5] described a tabetic with ocular and laryngeal crises who also exhibited paroxysmal attacks of salivation. A few years later Féré[6] observed a taboparetic with epileptic attacks involving the right side of the face and the right arm, in whom each attack was preceded by abundant salivation. Mazataud[7] was able to report four cases of salivation in tabes. His first patient exhibited a simple sialorrhœa. In the second patient the salivary crisis was announced by a feeling of nausea. The patient would then expectorate three or four large mouthfuls of saliva; with this he complained of a bitter taste in his mouth as well as of hallucinations of sight and hearing. Taste and smell were otherwise normal. The crisis lasted from one to three minutes only, but recurred once to five times a day. A third patient during attacks of gastric crises would experience a bitter taste in his mouth which was followed by sudden salivation lasting from one to two minutes, at the end of which time not only the salivation, but the gastric crisis as well, would cease. These attacks occurred ten times a day. The fourth patient had continuous salivation day and night; with this there was neuralgia of the left side of the face. The salivation continued for over a year, paroxysms occurring during which there was a sudden increase of secretion of saliva.

Klippel[8, 9] reports a case with autopsy findings. The patient complained of a continuous bad taste in his mouth which he likened to the taste of decayed fish. He exhibited changes in sensibility of the 5th nerve, on the face and lips, anæsthesia, formication, and flushing of the cheeks. On autopsy the salivary glands were large and much congested. In the parotid gland there were areas of round-cell infiltration, and the cells of the glandular acini were increased in

number and smaller than normal, with large nuclei. There was a catarrhal inflammation of the larger excretory ducts. In the submaxillary gland some of the acini appeared active and some inactive. He also found degenerative changes in the ganglia of Andersch and Gasser and in the 5th and 9th nerves. Klippel comments on the association of disorders of taste and smell, sensory disorders in the area of the trigeminal, tachycardia, and salivation.

André's[10] patient had salivary crises lasting from two to three hours in which the flow from the left side was greater than from the right. The patient also had attacks of left-sided neuralgic pains in the face, as well as hypæsthesia of the left side of the face and tongue. Taste was normal.

Umber[11] described a patient who experienced intensely disgusting tastes and smells, associated with a sensation of swelling in the neck and throat and a feeling of swelling of the submaxillary glands, with profuse salivation. These symptoms always came at the end of the gastric crisis. In another of his cases the gastric crises were preceded by great salivation and a very bad taste in the mouth. In both of these cases the salivation was apparently secondary to the disturbance of taste.

Alexander[12] in 1911 described a tabetic who had attacks of burning in the mouth with salivation, as well as a bad taste. They never occurred at meals. Taste and smell were otherwise normal. The saliva was thin and watery.

III.—CASE REPORT.

The following is a report of a case of syphilis of the nervous system with salivary crises, which we have studied at Montefiore Hospital.

The patient is a married man, 35 years of age, a furrier by occupation. He entered the hospital complaining of pain in the front of the upper left chest, of loss of weight, and of weakness. He has had no illnesses, with the exception of gonorrhœa at the age of 14 and a chancre at the age of 18. He received antiluetic treatment for four weeks. He states that at this time he lost his hair in ' bunches '. Twenty months ago, while rising from bed one morning, he was suddenly seized with pain behind the suprasternal notch, which radiated to the upper part of the left chest. He describes the pain as in the chest and as a choking, pressing sensation. He then vomited three or four times. Several days later he became dizzy and fell to the floor. For the first four months these attacks of pain occurred about every three weeks. Gradually the intervals became shorter, and during the last nine months the attacks of pain and vomiting have occurred almost daily. Their duration is variable, but they usually last a whole morning, and sometimes extend over several days. The patient was first treated at a gastro-intestinal clinic until a blood examination revealed a + + + + Wassermann. This test has been taken at several hospitals. At Bellevue it was + + + +,

at Mt. Sinai + + + +, at Harlem +, and at Montefiore it has twice been negative. After the first Wassermann was reported + + + + he received fourteen doses of salvarsan and twenty mercury injections, and the Wassermann became negative. However, his symptoms continued unabated. Three weeks after the treatment was instituted the patient began to complain of attacks of *severe salivation* lasting many hours. These have persisted, and at present occur almost daily. The salivation commences in the morning and is followed by retching, vomiting, and pain in the upper chest. Food is rarely seen in the vomitus. The salivation and pain last from four to twelve hours and occasionally persist over several days. With each attack the patient feels dizzy, and must lie down or hold on to something for fear of falling. The dizzy spells, however, last only a few seconds. The patient has lost twenty pounds in weight in the last year and a half. There was very slight difficulty of urination some months ago, but this has entirely disappeared. He never suffered from incontinence.

Physical Examination.—A man of small stature, weighing ninety-six pounds. Pale, rather poorly developed, and of normal intelligence. Teeth, tonsils, and pharynx in good condition. No evidence of gingivitis such as is seen in mercurial poisoning. Tongue slightly coated. Heart and lungs normal on physical examination. No increase in the area of aortic dullness, although the *x*-ray shows a slight widening of the aortic shadow. Electrocardiogram normal. Pulse-rate continuously rapid, ranging about 100 even when the patient is at rest. Efforts to take the pulse-rate while the patient is asleep have been unsuccessful, because he is such a very light sleeper. Blood-pressure is 135/75. Abdominal viscera normal. Gastro-intestinal *x*-ray negative. Gastric analysis shows the following :—

Fasting contents	145	c.c.
Free HCl	84	
Total acidity	91	
Ewald test meal	150	c.c. withdrawn
Free HCl	45	
Total acidity	55	

This indicates hypersecretion.

The spinal-fluid Wassermann was + while the patient was at Harlem Hospital. At Montefiore it was ±. The colloidal gold at Harlem Hospital was 0133100000, while at Montefiore it was 0111000000. The cell-count after a recent lumbar puncture showed 7 cells, globulin positive.

Neurological Examination.—Pupils are irregular ; left larger than right. No reaction to light, but good reaction with accommodation. Extra-ocular movements normal. No changes in functions of the 5th and 7th nerves. Pharyngeal reflex markedly diminished. Laryngeal reflex absent. No objective disorder of taste or smell. No abnormalities of the vocal cords. No atrophy or fibrillary twitchings of the tongue. *Deep reflexes all very active.* No Babinski or clonus. Abdominal reflexes present on both sides, and active. Muscle, joint, and vibration senses are normal. Pain on deep pressure over the calves, and Achilles tendon elicits a normal response. There is an approximately circular area of slight hypalgesia over the upper portions of the front of the chest. This is not always present. There are no sensory changes on the back. No ataxia and no Romberg. Gait is normal.

Progress.—During his stay in the hospital the patient has had attacks almost daily. His chief complaint is salivation, which, he says, induces the vomiting and which has been followed by the pain in the chest. At night his pillow has often been soaked with saliva, which drools from his mouth while he is asleep. During the attacks, as well as during the intervals, there are no sensory disorders of the face, mouth, tongue, neck, or pectoral regions. With the salivation the patient experienced a bad taste in his mouth, but there has been no objective disorder of taste or smell.

The following is a description of a typical attack.

Jan. 14, 1922.—This morning, on rising, the patient complained of burning in his throat, passing down to his chest. After dinner he began to complain of salivation and severe pain behind the suprasternal notch, passing downward and to the left. He then became nauseated, and vomited once. On examination 1½ hours after the onset, the patient was still in severe pain. He did not remain in one position, but moved about on the bed and rubbed his hand over his upper chest. At frequent intervals he expectorated a thin saliva. Walking did not aggravate the pain. No characteristic posture. Sublingual glands large. Their openings were prominent, red, and wide open, and saliva could be seen to flow from them. Pulse 126. Blood-pressure on several measurements 200/140 to 220/140, sitting. Heart and lungs showed no abnormality. No abdominal tenderness. Over the sternum and for several centimetres on either side there was a dull-red blotchy appearance, due to the injection of minute telangiectatic vessels. This disappeared when the clothes were removed. Atropine gr. $\frac{1}{60}$ given by hypodermic injection. Half an hour later the mouth became dry and salivation stopped. Pain worse.

Feb. 15, 1922.—The attacks have become more frequent and in the last month have occurred almost daily. Salivation took place almost every night. When asleep, saliva often drooled from his mouth, wetting the pillow and awakening him. On rising in the morning he has usually vomited and the salivation has become worse. If he had not salivated the night before, he did not vomit in the morning. After vomiting, the pain commenced behind the suprasternal notch and radiated to the left. The pain was usually not acute, but very uncomfortable, and was oppressive in character. It has been difficult to draw a deep breath. Nothing but morphine relieved this. When pain has been present, urination became difficult. The attack usually passed away at noon, and in the afternoon he felt well. Sometimes the attack persisted for several days. During the attack he cannot eat or drink.

During one of the intervals the patient was given $\frac{1}{5}$ gr. of pilocarpine hypodermically. This induced a very profuse salivation lasting about half an hour, as well as marked perspiration of the whole body, and abdominal cramps. No pain in the chest, nausea, or vomiting appeared. The saliva could be seen running from the parotid and sublingual ducts. The blood-pressure fell from 130/90 to 100/65. The patient has received no salvarsan or mercury for about six weeks, and shows absolutely no evidences of mercurialization.

IV.—DISCUSSION.

This patient does not present any of the signs of tabes dorsalis, but rather the picture of syphilitic cerebrospinal meningomyelitis.

In addition there are the following signs and symptoms:—(1) A profuse secretion of thin and watery saliva; (2) Gastric hypersecretion; (3) Attacks of vomiting related to salivation; (4) Tachycardia; (5) Attacks of high blood-pressure; (6) A sensation of bad taste in his mouth; (7) Pain in the suprasternal notch radiating downward and to the left; (8) Diminution of pharyngeal and absence of laryngeal reflexes.

Site of the Lesion.—All of these signs and symptoms are referable to involvement of the 7th, 9th, and 10th cranial nerves. If we assume that the involvement lies within the pons and medulla it is

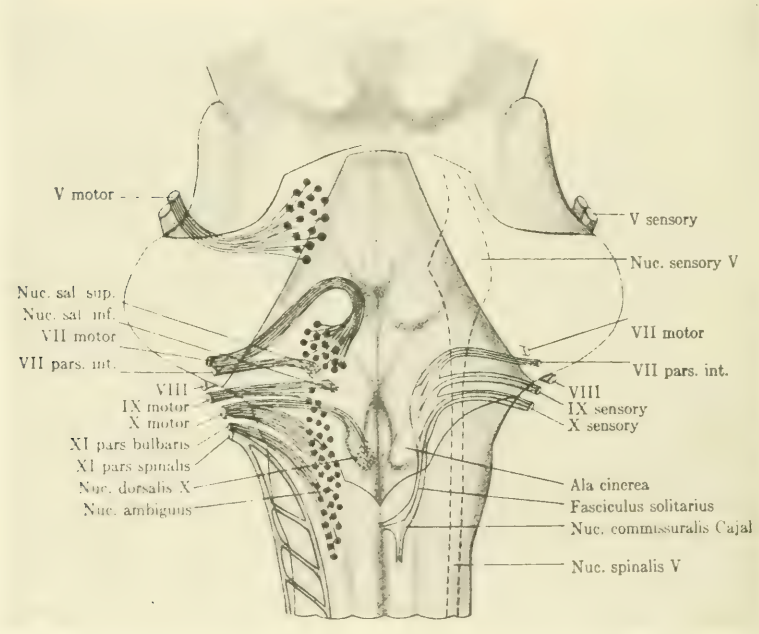

FIG. 1.—Diagram of the visceral afferent and efferent connections in the medulla oblongata. The afferent roots and centres are indicated on the right side; the efferent on the left. Visceral sensory fibres enter by the VII nerve (pars intermedia of Wrisberg, *VII pars. int.*) and by the IX and X nerves. These root-fibres include both general visceral sensory and gustatory fibres, all of which enter the fasciculus solitarius. (Fibres of the IX and X nerves also enter the spinal V tract; but since these are somatic sensory fibres from the auricular branch they are not included in the diagram).

On the left side of the figure the general visceral efferent nuclei are indicated by small dots, and the special visceral nuclei by large dots. The latter comprise the motor V nucleus for the jaw muscles, the motor VII nucleus for the muscles related to the hyoid bone and the general facial musculature, and the nucleus ambiguus supplying striated muscles of the pharynx and larynx by way of the IX and X nerves. Three general visceral efferent nuclei are indicated—the dorsal motor nucleus of the vagus under the ala cinerea and the superior and inferior salivatory nuclei. The superior nucleus (*nuc. sal. sup.*) supplies the sublingual and submaxillary salivary glands by way of the VII nerve (pars intermedia of Wrisberg), and the inferior nucleus (*nuc. sal. inf.*) supplies the parotid salivary gland by way of the IX nerve. All of the general visceral efferent fibres are preganglionic sympathetic fibres which end in sympathetic ganglia, whence postganglionic fibres carry the nervous impulses onward to their respective destinations.

(From *An Introduction to Neurology*, C. J. Herrick, 1916, p. 156.)

possible to account for all the findings. Involvement of the dorsally placed visceral vagus nucleus (tachycardia, vomiting, gastric hypersecretion), involvement of the adjacent sensory vagus nucleus (pain), of the fasciculus solitarius (taste), the nuclei salivatorii of Kohnstamm (salivation), and the glossopharyngeal and vagus nuclei (diminution of pharyngeal and absence of laryngeal reflexes), account for everything that the patient presents. It is possible that this group of symptoms and signs is produced by neuritis of the glossopharyngeal and vagus nerves. Without post-mortem examination it is impossible to be absolutely certain which of these hypotheses is correct. We incline to the idea that the process is central.

The nervous control of salivation by the autonomic originates in the nuclei of Kohnstamm which lie near the 7th and 9th nuclei.

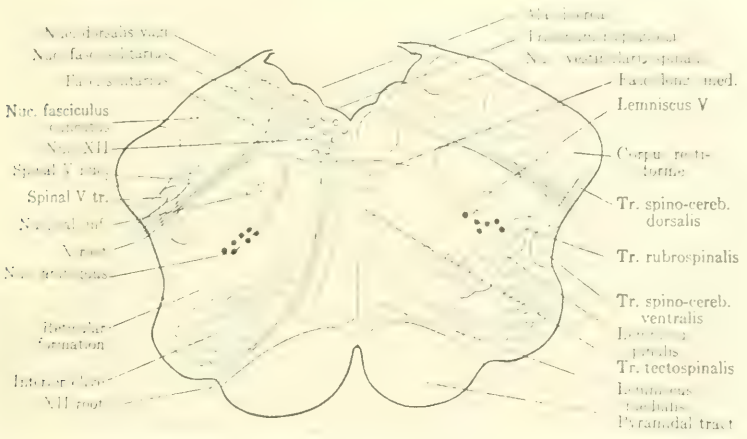

FIG. 2.—Diagrammatic cross-section through the human medulla oblongata at the level of the vagus nerve, illustrating details of functional localization.

(From *An Introduction to Neurology*, C. J. Herrick, 1916, p. 244.)

The position of the nuclei and the course of their fibres is indicated in the accompanying charts (*Figs.* 1, 2, and 3). The fibres controlling the stomach pass via the vagus and account for the gastric hypersecretion and vomiting. Except at the onset of the disease, the vomiting has always followed an attack of profuse salivation, and might be thought to be due to the filling of the stomach with a large amount of swallowed saliva. In view of the onset with gastric symptoms *unassociated* with salivation and the evidence of hypersecretion by gastric analysis, we feel that a secretion similar to that of the saliva occurs in the stomach. This and the vomiting are therefore separate manifestations, and occur more or less parallel to the salivation.

The tachycardia and the attacks of high blood-pressure are not easy to explain. Much has been written emphasizing the relation of high blood-pressure to gastric crises. However, from a recent piece of work at Dejerine's clinic by Heitz and Norero[13] it is evident that no close relationship exists between these two manifestations. The rise in blood-pressure and the pain of gastric crises

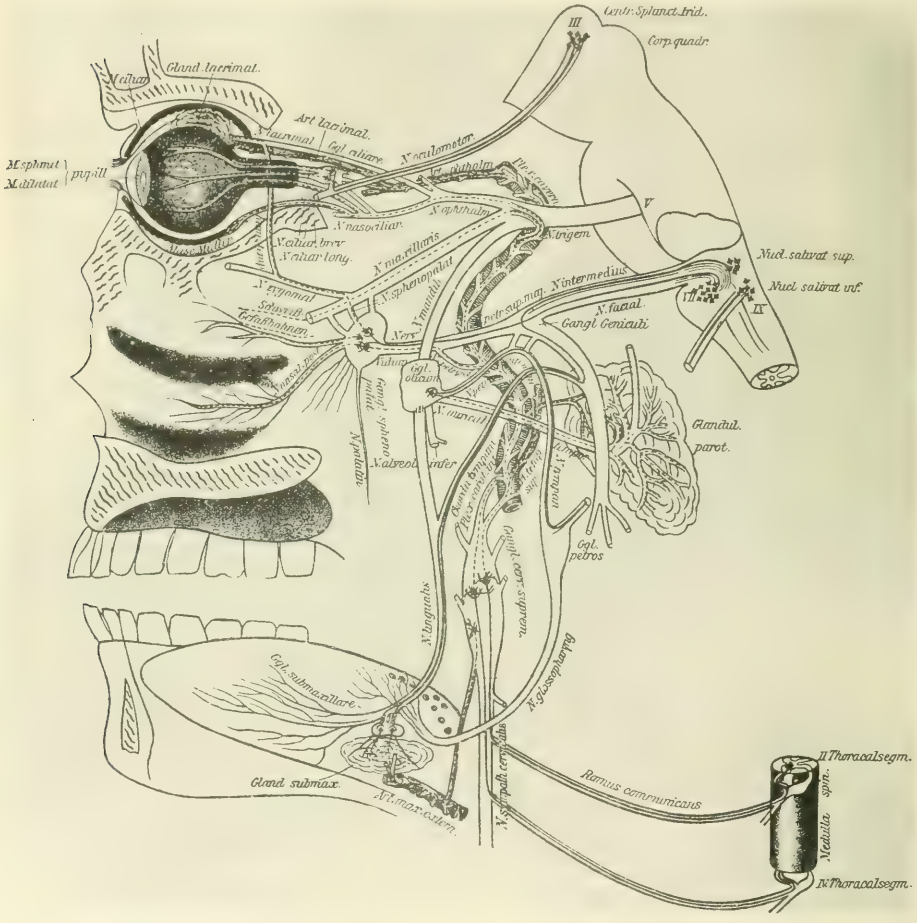

Fig. 63.—Schematic representation of the vegetative nerve innervation of the head. (Müller and Dahl.)

(From *Vegetative Neurology*, Heinrich Higier, 1919, p. 19. Nervous and Mental Diseases Monograph Series, No. 27. Translated by Walter M. Kraus.)

appear to be separate manifestations dependent upon a common cause rather than causally related to one another. There never was any abdominal pain or tenderness during the patient's attacks. However, pain behind the suprasternal notch has been present. In spite

of this fact, a rise of blood-pressure had not existed during all the attacks of pain, emphasizing the lack of any relationship. The cases reported in the literature include many of the signs and symptoms which we have described. In addition, associated involvement of the 5th nerve has been reported several times.

V.—CONCLUSIONS.

1. Crises occur in patients with syphilis of the nervous system who do not present the picture of tabes dorsalis.

2. A group of signs and symptoms due to disease of the pons and medulla or their nerves occurs in such cases of syphilis of the nervous system, and includes: (*a*) A profuse secretion of thin and watery saliva; (*b*) Gastric hypersecretion; (*c*) Attacks of vomiting related to salivation; (*d*) Tachycardia; (*e*) Attacks of high blood-pressure; (*f*) A sensation of bad taste in the mouth; (*g*) Pain in the suprasternal notch radiating downward to the left; (*h*) Diminution of pharyngeal and absence of laryngeal reflexes; (*i*) Disorders of sensation in the trigeminal distribution.

3. Whether the entire picture described be due to central or peripheral involvement remains to be decided. We incline to the former point of veiw.

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THE DIFFERENCE BETWEEN A MUSCULAR AND A NEUROMUSCULAR INTERPRETATION OF WALKING*

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NEW YORK

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I. Introduction

Walking is a highly complicated process. The underlying mechanisms, in view of their importance in all studies of motility, have attracted attention at least since the days of the Weber brothers, nearly one hundred years ago. The general conception of the movements of the four extremities has not changed essentially since it was first described. These movements were recently described thus: "The right upper and left lower limbs are coupled together and swing forwards or backwards at the same time, flexion of one arm occurring with extension of the leg on the same side."[1] In other words, both the homolateral limbs as well as the pairs of arms and legs, respectively, move in opposite directions. This conception is based on ordinary observations and on the action of certain definite muscles. Though it has been recognized since the days of Huxley that the front of the leg is, in reality, the dorsal surface and though this has been abundantly proved by various anatomists who have studied the comparative anatomy and embryology of the limbs, the significance of this has apparently attracted little attention among neurologists and has not been used in the interpretation of group movements, normal or abnormal, of man. The process of walking has therefore been discussed from the point of view of muscular action, on one hand, and

* Read by title at the Forty-Eighth Annual Meeting of the American Neurological Association, May, 1922, Washington, D. C.

1. Riddoch, George, and Buzzard, E. Farquhar: Reflex Movements and Postural Reactions in Quadriplegia and Hemiplegia, with Especial Reference to Those of the Upper Limb, Brain **44**:462, 1921.

from the point of view of its general control as a reflex mechanism by the spinal cord on the other. The classical work of Sherrington has established definite laws for this reflex action and has also indicated which muscles are involved in certain phases of reflex stepping (Fig. 1, Table 1). However, in all this, no attempt seems to have been made to interpret walking from a point of view which considers the embryology and evolutionary origin of the muscles involved. I shall attempt to show that the spinal cord controls, by its integrations, large groups of muscles which are related, not only embryologically, but also by virtue of their common nerve supply. On this basis it may be shown that the integrations controlling walking are among the foundations of the nervous system and constitute primitive motility patterns. All

TABLE 1.—The Muscles Shown in Figure 1 Arranged in Ventral and Dorsal Groups *

	A		B	
	Ventral	Dorsal	Ventral	Dorsal
I.	.....	I. Psoas M. Gluteus minimus R. Rectus femoris F. Tensor fasciae femoris brevis S. Sartorius lateralis S'. Sartorius medialis	0. Quadratus femoris 3. Adductor minor 4. Adductor major (a part) 5. Semimembranosus 6. Biceps femoris posterior	
II.	B. Biceps femoris posterior G. Gracilis T. Semitendinosus	.....	.....	1. Crureus 2. Vasti
III.	.....	A. Tibialis anticus L. Extensor longus digitorum P. Peroneus longus E. Extensor digitorum brevis	7. Gastrocnemius 8. Soleus 9. Flexor longus digitorum	

* The first group in both tables includes muscles acting at the hip, the second those acting at the knee, the third those acting at the ankle.

the neurons engaged in producing patterns necessary to gait are of spinal origin. On these are superimposed other patterns appearing later in the course of development and more complicated mechanically.

In order to appreciate the significance of the various integrations controlling motility, it is necessary first to understand the most primitive of these and the manner in which they control muscles. Emerging from this method of interpretation of movement—which has been designated the neuromuscular [2] and which considers the grouping of the peripheral motor neurons and their end-organs, the muscles—the idea

2. Kraus, Walter M.: A Principle Hitherto Undescribed of the Physiology of Movement and Posture. The Primitive Integration of Movement in Vertebrates, Arch. Neurol. & Psychiat. **7**:381 (March) 1922.

will be developed that though the actual visible movements involved in walking indicate that in reality the homolateral limbs move in opposite directions, when their control by the nervous system is considered, these opposite movements are equivalent to movement in the same direction. Furthermore, it will be shown that there exist corresponding opposite phases of gait in which the analogues of the activity of singly hinged appendages in primitive animals can be made out, and

Fig. 1.—Reproduced from *Brain*, volume 33, page 22, 1910, from the article by C. S. Sherrington on "Remarks on the Reflex Mechanisms of the Step."

that superimposed on these is another pair of corresponding opposites also controlled by the spinal cord and based on the development, in the course of evolution, of the three-hinged appendages of the land-living vertebrate. Such an analysis of the control of walking by spinal integrations demands an entirely new point of view toward posture and progression patterns, not only in normal man but also in all disorders of the central nervous system in which these neurons are disturbed.

II. THE ANATOMIC AND PHYSIOLOGIC BASES OF THE NEUROMUSCULAR MECHANISM

As has been said before,[3] movements are now described in such terms as extension, abduction, internal rotation, etc. In other words, we describe the activity of a neuromuscular mechanism in terms of the muscular part of that mechanism alone, disregarding the neurologic element of movement and its control by the spinal cord and its peripheral nerves. The neural part of the mechanism is represented by peripheral motor neurons. The activation of groups of these neurons in various ways by the spinal cord is carried out by neurons within the spinal cord and is known as integration. When we describe in terms of muscular action only, complicated movements guided by integrations of the nervous system at levels limited to or higher than the spinal cord,

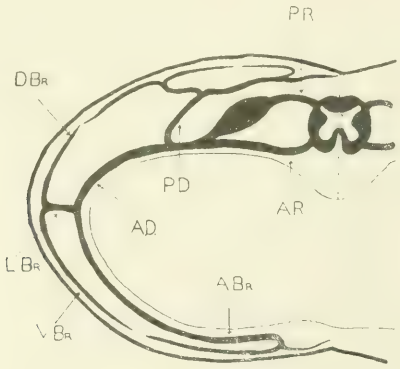

Fig. 2.—Adapted from "Human Anatomy" by G. A. Piersol, Part 2, p. 1284, Fig. 1085. *D. Br.*, dorsal branch; *V. Br.*, ventral branch; *L. Br.*, lateral branch; *A. Br.*, anterior branch; *P. R.*, posterior root; *A. R.*, anterior root; *P. D.*, posterior division; *A. D.*, anterior division.

we omit consideration of the spinal patterns and control of the final neuromuscular mechanism.

The nervous system is unquestionably built up of a series of physiologic levels of which the higher or more recently developed, phylogenetically speaking, carry out functions which are different from the lower. As Herbert Spencer remarked, "Integration keeps pace with differentiation." As animals become more and more differentiated, more and more needful of performing new kinds of movements in pattern, integrations appear to carry out these group movements. It is my opinion that we must have some conception of the integrating action of the lowest levels in order to interpret the higher. We must know how the spinal cord integrates movements in order to understand the activities and patterns of higher integrations superimposed in the course of

evolution and ultimately to understand why it has become necessary for these higher integrations to appear at all. It is necessary to consider in detail the general plan of the peripheral nerves, the general plan of the innervation of muscles by them and the embryologic grouping of the muscles themselves in order properly to examine integration of movement by the spinal cord.

A typical thoracic spinal nerve having nothing to do with the innervation of limbs shows a number of divisions after union of the anterior and posterior spinal roots. The trunk so formed divides into an anterior and posterior division. The anterior division divides further into a lateral and an anterior branch, the lateral branch dividing into a dorsal

TABLE 2.—Division of the Nerves of the Extremities into Dorsal and Ventral Groups

Origin	Groups	Nerves
		Upper Extremity
Brachial plexus	Dorsal trunks (posterior cord)	Dorsal scapular Long thoracic Suprascapular Subscapular (2) Thoracodorsal Axillary Radial
	Ventral trunks (lateral and medial cords)	Nerve to subclavius Anterior thoracic (2) Musculocutaneous Median Ulnar
		Lower Extremity
Lumbosacral plexus	Dorsal trunks	Superior gluteal Inferior gluteal Nerve to piriformis Femoral Peroneal
	Ventral trunks	Obturator Nerve to obturator internus and superior gemellus Nerve to quadratus femoris and inferior gemellus Tibial

and a ventral (Fig. 2). As far as the axial musculature is concerned, the portions of it developed from the ventrolateral portions of the embryo are supplied by the anterior division and its branches, while the dorsal musculature is supplied by the posterior branches. Thus, a simple scheme results from a physiologic point of view. There emerges the notion that, in general, movements of extension are guided by the posterior division, while flexion is guided by the anterior division. The lateral and rotatory movements produced by these groups of muscles are due to overactivity of the right and left halves of either the dorsal or the ventral groups or both. One theory assumes that the limb plexuses are made up only of the lateral branch of this division. For the purposes of this discussion, it is not important to try to reach a decision as to which of these theories is correct.

The primitive extremities of the embryo are flattened buds having a dorsal and ventral surface. The "premuscle mass" on the dorsal side gives rise to muscles the majority of which, in the adult, have an extensor function. The "premuscle mass" on the ventral side gives rise to muscles the majority of which have a flexor function. The

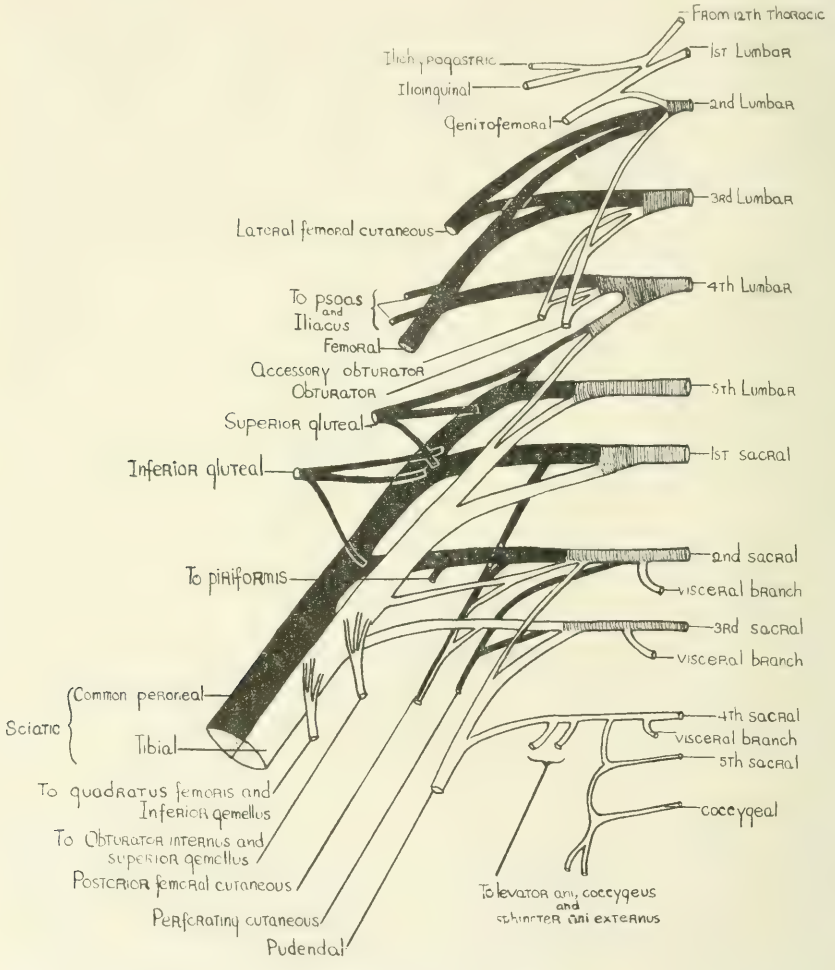

Fig 3.—Plan of the lumbar, sacral and pudendal plexuses. Black, dorsal; white, ventral; cross-hatched, combined. (Modified from Gray's "Anatomy," pp. 949 and 958).

crux of the whole situation lies in the fact that certain muscles have a function opposite to that which would be expected from a knowledge of the side of the limb bud (ventral or dorsal) from which they develop. For example, the iliopsoas group develops from the dorsal

muscle mass of the lower limb bud, but has a ventral function, that is, flexion.[2] This is an old and well-known fact, and is due to rotation of the leg in the course of evolution.

To return now to the question of innervation, we find that the embryology of the muscles is beautifully emphasized by their innervation. It is well known that the nerves of the brachial and lumbosacral plexuses divide into two definite groups, one of which is posterior or dorsal and the other anterior or ventral. In the arm, the ventral branches of the brachial plexus innervate the subclavius and pectoral muscles as well as all the muscles supplied by the musculocutaneous, median and ulnar nerves. The remaining nerves are dorsal (Table 2). The same division in the lumbosacral plexus is indicated by Figure 3 and Table 2.

A detailed table of muscles based on their derivation from the dorsal or ventral aspects of the limb bud and their supply by the dorsal or ventral branches of the limb plexuses shows that those muscles which develop from the dorsal layers of the bud are supplied by the dorsal branches of the limb plexus and those which develop from the ventral layers by the ventral branches. The analogy to the condition in the axial musculature is obvious (Tables 2, 3, 4 and 5).[3]

From all this we arrive at some possibility of schematization. In the nerves and muscles of the upper and lower limbs we find, as in the case of the iliopsoas mentioned before, that muscles which would be expected to have an extensor action have in reality a flexor action and vice versa. In other words, were we to group the muscles acting in any given movement merely on the basis of their activity as muscles and leave out of consideration their embryology and activating nerves, we would be leaving out entirely the division of these nerves and of their end-organs, the muscles, into large ventral and dorsal groups.

To illustrate how this information may be used for discovering just how the spinal cord may integrate movement, let us consider the use of relatively simple spinal integrations in the process of walking.

III. TECHNIC USED IN ANALYZING THE PROCESS OF WALKING

Simple observation of walking and running in a normal man does not permit of a precise analysis of the various phases of gait. The movements are too rapid and the transitions between flexion and extension, etc., are too small to give accurate results. Advantage was there-

3. Lewandowsky, M.: Handbuch der Neurologie. Flatau, Edward: Die Motorische, Sensible und Reflex Segmentierung im Rückenmark, Allg. Neurol. **1**, Pt. 2, p. 659, 1910. Cunningham, D. J.: Textbook of Anatomy, New York, William Wood & Co., 1918, p. 743. Braus, Hermann: Anatomie des Menschen, Bewegungsapparat, Berlin **1**: 1921.

fore taken of cinematographic pictures by which a considerable number of successive phases of walking and running could be obtained.

The pictures taken were a series from X to Z (Fig. 4). X Y and Z were indicated on a black drop by sheets of white paper. Y indicates

TABLE 3.—Division of the Muscles of the Upper Extremity into Dorsal and Ventral Groups

	Dorsal		Ventral
1	Levator scapulae	1	
2	Serratus anterior	2	
3	Rhomboideus major	3	
4	Rhomboideus minor	4	
5	Supraspinatus	5	
6	Infraspinatus	6	
7	Teres minor	7	
8	Deltoid	8	
9	Subscapularis	9	
10	Teres major	10	
11	Latissimus dorsi	11	
12		12	Subclavius
13		13	Pectoralis major
14		14	Pectoralis minor
15		15	Biceps brachii
16		16	Brachialis *
17		17	Coracobrachialis
18	Brachioradialis	18	
19	Extensor carpi radialis longus	19	
20	Extensor carpi radialis brevis	20	
21	Supinator	21	
22	Extensor pollicis longus	22	
23	Extensor indicis proprius	23	
24	Abductor pollicis longus	24	
25	Extensor pollicis brevis	25	
26	Extensor communis digitorum	26	
27	Extensor carpi ulnaris	27	
28	Extensor minimi digiti quinti	28	
29	Anconeus	29	
30	Triceps	30	
31		31	Pronator teres
32		32	Flexor carpi radialis
33		33	Palmaris longus
34		34	Flexor digitorum sublimis
35		35	Flexor digitorum profundus
36		36	Flexor pollicis longus
37		37	Pronator quadratus
38		38	Lumbricalis 1
39		39	Lumbricalis 2
40		40	Lumbricalis 3
41		41	Lumbricalis 4
42		42	Abductor pollicis brevis
43		43	Opponens pollicis
44		44	Flexor pollicis brevis (lateral head)
45		45	Flexor pollicis brevis (medial head)
46		46	Flexor carpi ulnaris
47		47	Adductor pollicis obliquus
48		48	Adductor pollicis transversus
49		49	Interosseus volaris 1
50		50	Interosseus volaris 2
51		51	Interosseus volaris 3
52		52	Interosseus dorsalis 1
53		53	Interosseus dorsalis 2
54		54	Interosseus dorsalis 3
55		55	Interosseus dorsalis 4
56		56	Opponens digiti quinti
57		57	Flexor digiti quinti brevis
58		58	Abductor digiti quinti

* Supplied by both dorsal and ventral nerves.

the point at which the subject was directly in front of the camera and X and Z two points equidistant from Y on either side. The distance X—Z is equal to 17 feet. In order to include positions X and Z, a panoramic series was used. Of the series reproduced, seventeen pictures were

TABLE 4.—Division of the Muscles of the Lower Extremity into Dorsal and Ventral Groups

	Dorsal		Ventral
1	Iliacus	1	
2	Psoas major	2	
3	Psoas minor	3	
4	Pectineus *	4	
5	Sartorius	5	
6	Rectus femoris	6	
7	Vastus lateralis	7	
8	Vastus medialis	8	
9	Vastus intermedius	9	
10	Tensor fasciae latae	10	
11	Gluteus minimus	11	
12	Gluteus medius	12	
13	Piriformis	13	
14	Gluteus maximus	14	
15	Biceps femoris, short head	15	
16		16	Biceps femoris, long head
17		17	Semitendinosus
18		18	Semimembranosus
19		19	Adductor magnus
20		20	Obturator externus
21		21	Adductor longus
22		22	Adductor brevis
23		23	Gracilis
24		24	Obturator internus
25		25	Superior gemellus
26		26	Interior gemellus
27		27	Quadratus femoris
28	Tibialis anterior	28	
29	Extensor hallucis longus	29	
30	Extensor digitorum longus	30	
31	Peroneus tertius	31	
32	Peroneus longus	32	
33	Peroneus brevis	33	
34	Extensor digitorum brevis	34	
35		35	Gastrocnemius
36		36	Soleus
37		37	Plantaris
38		38	Popliteus
39		39	Tibialis posterior
40		40	Flexor digitorum longus
41		41	Flexor hallucis longus
42		42	Quadratus plantae
43		43	Abductor digiti quinti
44		44	Flexor digiti quinti brevis
45		45	Opponens digiti quinti
46		46	Plantar interosseus 1
47		47	Plantar interosseus 2
48		48	Plantar interosseus 3
49		49	Dorsal interosseus 1
50		50	Dorsal interosseus 2
51		51	Dorsal interosseus 3
52		52	Dorsal interosseus 4
53		53	Adductor hallucis
54		54	Lumbricalis 1
55		55	Lumbricalis 2
56		56	Lumbricalis 3
57		57	Lumbricalis 4
58		58	Abductor hallucis
59		59	Flexor digitorum brevis
60		60	Flexor hallucis brevis

* Supplied by both dorsal and ventral nerves.

used for analysis. It required three and one-fifth seconds to walk from *X* to *Z* and about one second to walk one pace. By a stroke of good luck the picture showing one leg in support and the other in maximum flexion (No. 25) was but one from *Y*, that is, almost directly in front of the camera (Figs. 4 and 5). The degree of rotation of the body appearing at *X* and *Z* is shown in Figure 6. This may be compared to Figure 5.

TABLE 5.—Division of the Muscles of the Axis into Dorsal and Ventral Groups

	Ventral	Dorsal
Muscles of the Back:		
Serratus posterior superior		+
Serratus posterior inferior		+
Splenius		+
Sacrospinalis		+
Iliocostalis		+
Longissimus		+
Spinalis dorsi		+
Semispinalis		+
Multifidus		+
Obliquus capitis inferior		+
Obliquus capitis superior		+
Rectus capitis posterior major		+
Rectus capitis posterior minor		+
Rotatores		+
Interspinales		+
Intertransversarii	+	
Trapezius		+
Muscles of the Neck:		
Scalenus anterior	+	
Scalenus medius	+	
Scalenus posterior	+	
Longus capitis	+	
Rectus capitis anterior	+	
Longus colli	+	
Rectus capitis lateralis	+	
Sterno-kleido-mastoid	+	
Muscles of the Thorax:		
Intercostales	+	
Levatores costarum	+	
Subcostales	+	
Transversus thoracis	+	
Muscles of the Abdominal Wall:		
Obliquus externus abdominis	+	
Obliquus internus abdominis	+	
Cremaster	+	
Transversus abdominis	+	
Pyramidalis abdominis	+	
Rectus abdominis	+	
Quadratus lumborum	+	
Muscles of the Perineum:		
Sphincter ani externus	+	
Corrugator cutis ani	+	
Transversus perinei superficialis	+	
Bulbocavernosus	+	
Ischiocavernosus	+	
Sphincter urethrae membranaceae	+	
Transversus perinei profundus	+	
Muscles of the Pelvis:		
Levator ani	+	
Coccygeus	+	

Except for careful mathematical analysis, this rotation is not important, though in all analyses it should be regarded as a source of error in drawing conclusions. Before taking the pictures it was determined that the difference between having the subject walk part of the circumference of a circle indicated by *X*, *W*, *Z* (Fig. 4), and walking a straight line, *X*, *Y*, *Z*, was so slight as to make its carrying out unimportant. This difference, the distance from *Y* to *W*, is only a foot and a half.

A series of cinematographic pictures was projected one after another on a wall at a distance of 12 feet. The images produced were 19 inches high. The method of determining the successive movements or changes in the angular relations of the thigh and torso, or of the knee and the thigh, was to trace a series of pictures with, in the first instance, the torso of each successive picture superimposed on the preceding one. The thigh movements are thus made out (Figs. 7 and 8). In tracing the thigh-knee relations, the thigh was taken as a constant

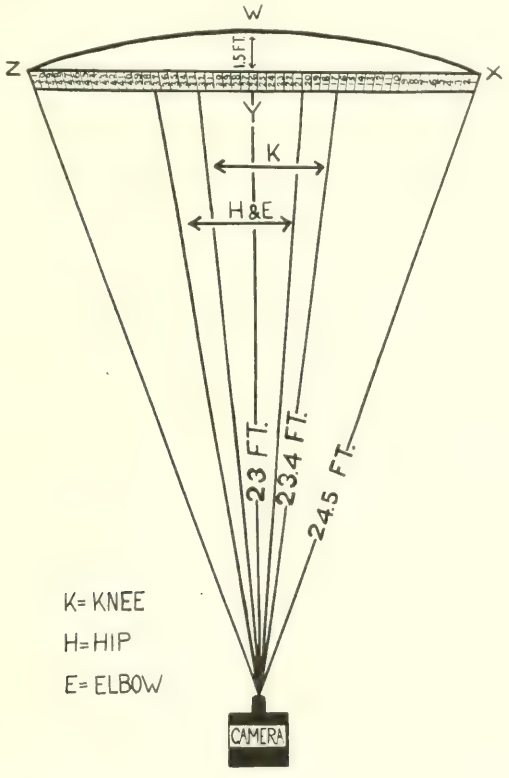

Fig. 4.—Diagram representing the distance relations of the patient to the camera. The patient walked a straight line from *X* to *Z*. The small numbers between *X* and *Z* each represent a picture. For further explanation see text, section III.

(Figs. 9 and 10). The elbow movements were determined by the same method (Fig. 11). Because of rotation, the shoulder movements cannot be illustrated easily and have been omitted. Certain precautions must be taken in such successive tracings in which changes in the positions of muscles cause changes in contour. The difference in the lower posterior thigh line, caused by changing positions of the hamstrings,

is illustrated in Figure 12, in that *A* shows the contour in flexion at the knee, *B* the contour in extension at the knee. In all drawings the anterior thigh line was used as the standard.

The horizontal has been inserted in all the pictures of the torso-thigh series. In the thigh-knee series the horizontal for the first tracing of each series has been inserted. A vertical line through the central position between ventral and dorsal and at right angles to the floor line was put into the torso-thigh cuts to indicate tilting of the torso and the relative change of the center of gravity in these positions.

It is obvious that a complete pace must be taken for analysis. It so happened that seventeen pictures were needed to show this. If,

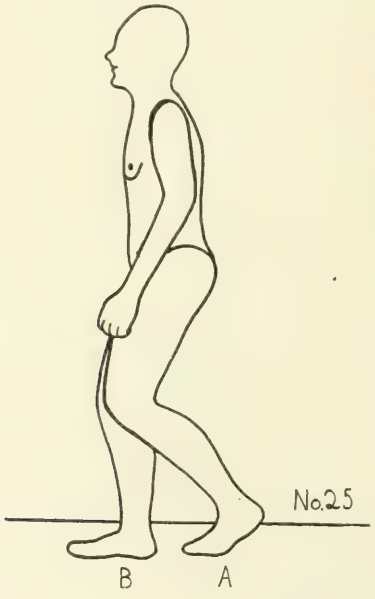

Fig. 5.—This tracing shows the left leg, A, in the maximum position of the flexion reflex and the right leg, *B*, in the maximum position of support. These constitute the third and fourth phases of walking. The actual picture, as well as the positions, are mathematically midway between those shown in Figures 13 and 14. The thighs are seen to be in alinement with one another. Flexion at the knee of the right leg, *B*, is apparent.

therefore, the first and seventeenth are the same, the ninth is the middle picture and must represent a stage midway between, and one in which the legs are in mirrored positions of those in the first and seventeenth. Furthermore, it would be expected that midway between the first and ninth and the ninth and seventeenth there would occur the maximum position of flexion reflex in one leg and a maximum position of support in the other. The analysis bore this out, and the fifth and thirteenth

represented these. To check this, they were compared. The arm positions were also used to determine the accuracy of the conclusions. When the arms are parallel to one another, one leg is in maximum support, *B*, and the other in maximum flexion, *A* (Fig. 5). An analysis of succeeding pictures furnishes a check to the selection of the various stages of the pace, irrespective of any suppositions on purely mathematical grounds. Still another method of determining by analysis of the knee movements that the fifth and thirteenth are indeed maximum positions of flexion reflex and support will be illustrated below. The actual pictures used were numbers 21 to 37 for the hip, shoulder and elbow and numbers 17 to 33 for the knee. A slightly

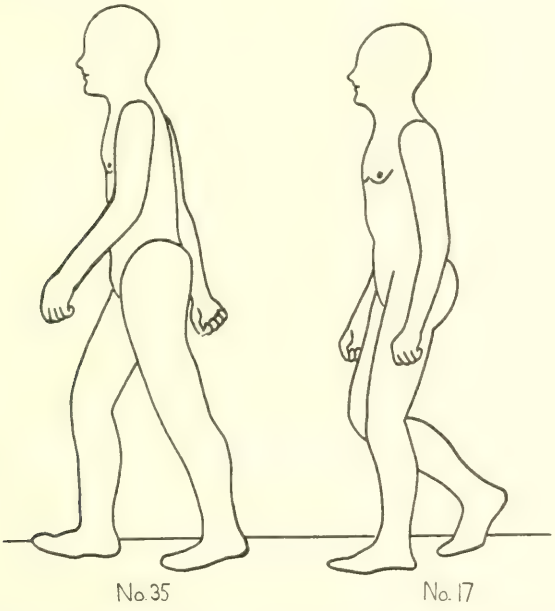

Fig. 6.—Rotation produced in a paroramic series of pictures, due to the patient's walking in a straight line. To avoid this rotation the patient should walk along the arc of a circle (Fig. 4, *X, W, Z*).

different series was taken for the knee in order to emphasize the difference in the extent of oscillation during the posture and flexion phases.

IV. MOVEMENTS DURING WALKING

For the purposes of the present analysis of the activity of the legs in walking, the assumption has been made that it is composed of four simple activities, namely: (1) the ventral, finlike movement of the lower limbs at the hip; (2) the dorsal, finlike movement of the lower limbs at the hip; (3) the flexion reflex element, and (4) the support element.

Finlike Movement at the Hip.—For the moment let us omit the last two elements. If the assumption concerning these four activities be true, one may expect that in the process of walking the leg undergoes a pendulum-like action from the points of attachment of the leg at the transervse plane of the hip. Such an action was postulated in 1836 by the brothers Weber, though they attributed this activity not to muscular but to gravity effects. Both Vierordt and Duchenne of Boulogne have considered that this activity is a part of the process of walking, carried out not by gravity, but by muscles. In an analysis of a complete pace of a normal person by means of tracings of successive cinematographic pictures, it has been found that in the process of walking the movement at the hip is continuous (Figs. 7 and 8).

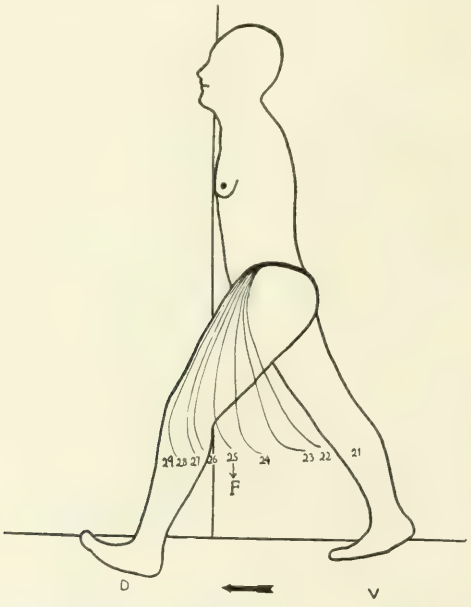

Fig. 7.—Consecutive tracings of the movement of the thigh in its progress from the ventral to the dorsal position. Compare with Figure 8.

It is well recognized now that the singly-hinged appendages of the fish preceded the three-jointed appendages of land vertebrates, such as amphibia. The most primitive grouping of movements by the spinal cord in the process of progression must have been one which correlated the activities of muscles much simpler than our own which lay on the ventral and dorsal aspects of the appendages, respectively. It seems obvious that the two groups of muscles which carried the fins in opposite directions must be controlled by spinal neurons permitting reciprocal innervation, so that when an agonist contracts its antagonist

is relatively relaxed. In order to realize this primitive finlike movement of a singly hinged appendage in man, it is necessary to regard the human leg as a rod and to ascertain whether there is a stage in the process of walking at which it assumes either a forward (dorsal) or backward (ventral) position. It is also necessary to determine whether these positions occur at regular intervals and whether they appear in definite relation to the two other elements of gait described by Sherrington as the extensor and flexor phases (Fig. 1).

It has been found that these two ventral and dorsal positions of either leg regarded as a rod do occur at regular intervals in the course of walking (Figs. 13 and 14), and it is found, furthermore, that

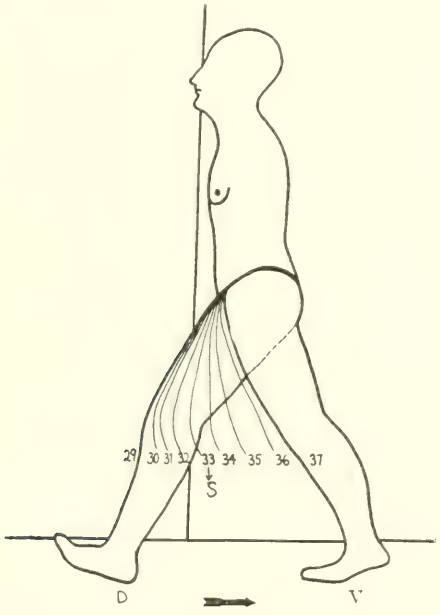

Fig. 8.—Consecutive tracings of the movement of the thigh in its progress from the dorsal to the ventral position. Compare with Figure 7.

midway between the appearance of the ventral and dorsal positions the flexion reflex element occurs, while the support element occurs midway between the appearance of the dorsal and ventral positions. It seems apparent, therefore, that in the process of walking the two primitive finlike movements at the hips can be made out with ease. That they occur rhythmically, that they are diphasic, continuous and represent alternating movements of the dorsal and ventral groups of hip muscles is illustrated by Figures 7 and 8. The rodlike positions are seen only at two stages of gait and are concealed at all other stages, due to the addition of the support and flexion elements.

The Flexion Reflex Element.—Since these movements which have just been described are necessarily more primitive than those of the three-jointed limbs of the land animal, it is assumed that the elements in walking developed by the land animal are considerably more complex and do not represent simple pendulum-like activities. As a matter of fact, the flexion reflex element represents an alternation of movement at the three great joints. This is beautifully demonstrated by an examination of the muscles given by Sherrington [4] in his paper, "Remarks on the Reflex Mechanism of the Step," as those causing the extensor and flexor phases of gait. What Sherrington called the flexion phase of reflex stepping corresponds precisely to the third element of gait in this analysis. Figure 1 and Table 1, taken from Sherrington's paper, illustrate this. The table of muscles, to which there has been added the words "ventral" and "dorsal," indicates the alternation of movement—dorsal at the hip, ventral at the knee and dorsal at the ankle.

The Support Element.—The extensor phase which Sherrington described shows precisely the opposite picture. That omission of the neuromuscular mechanism may lead to an attempt physiologically to group movements obviously not susceptible to such grouping is by now apparent. Two examples of such an attempt appear in Walshe's [5] paper on "The Physiological Significance of the Reflex Phenomena in Spastic Paralysis of the Lower Limbs." He says:

> I have said above that all the flexors of the limb are activated in the flexion reflex. These include hip and knee flexors and the dorsiflexors of the ankle and digits. Now, of these, the dorsiflexors, in anatomical nomenclature, are named extensors. There can be no doubt, however, that these muscles must be grouped physiologically with the flexors. . . . In view of the fact that the "extensor response" is a part of a general movement of flexion while the toe movement in the normal plantar response, the "flexor response," is, physiologically considered, a movement of extension, it is unfortunate that the accepted terminology is the direct converse of the facts. The expressions "extensor response" and "flexor response" are widely used by neurologists in this country, and I have therefore been compelled to make use of them, though it is very difficult to prevent confusion in the mind of the reader when speaking of a "crossed extension reflex" and of a "crossed extensor response"; two very different phenomena involving antagonistic groups of muscles. I wish, therefore, to emphasize that these empirical and misleading names are used here in their accepted sense, and have no physiological connotation such as the terms "flexion reflex" and "crossed extension reflex" have. I have consequently placed them in inverted commas to avoid as far as possible all confusion.

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4. Sherrington, C. S.: Remarks on the Reflex Mechanism of the Step, Brain **33**:22, 1910-1911.

5. Walshe, F. M. R.: The Physiological Significance of the Reflex Phenomena in Spastic Paralysis of the Lower Limbs, Brain **37**:269, 1914-1915.

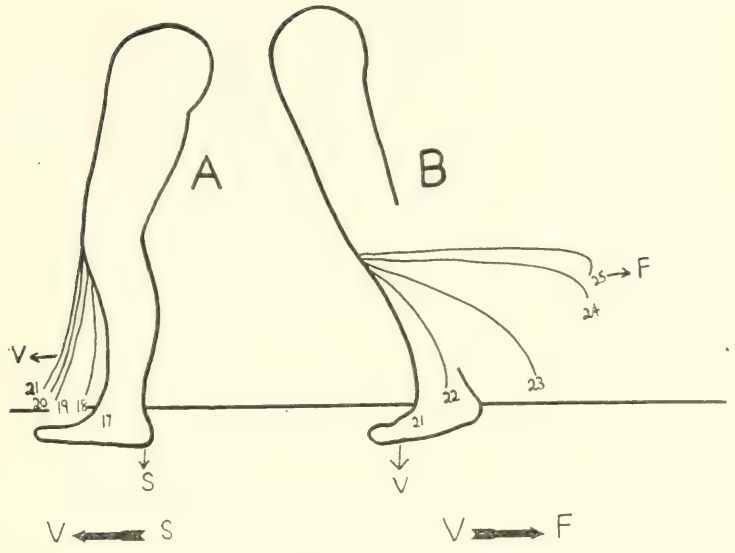

Figure 9

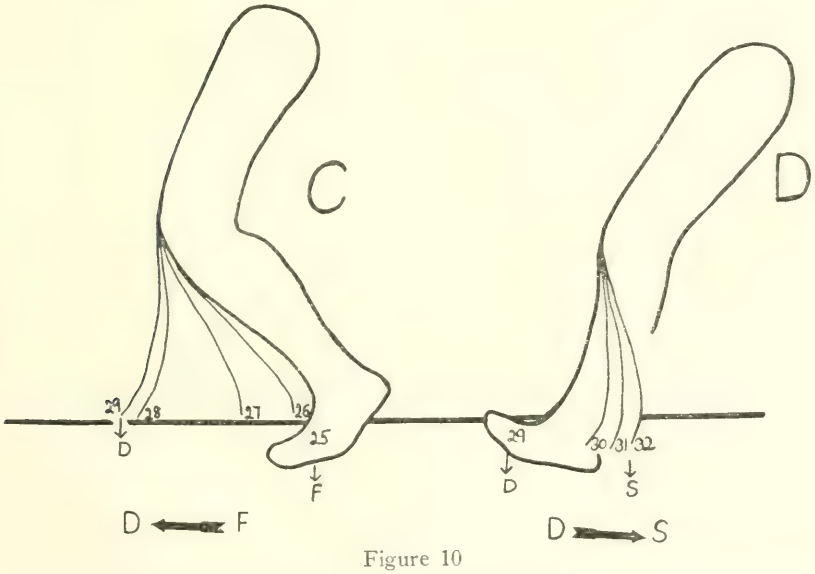

Figure 10

Figs. 9 and 10.—These illustrate the tetraphasic character of the knee movements. *A* and *D* constitute the diphasic posture element, *B* and *C* the diphasic flexion element. Only four tracings are shown in *D* as the next picture showed a position in the knee slightly in advance of its predecessor. This indicates a slight difference in the speed of walking or in the speed of taking the pictures. As a result only four pictures instead of five are needed to represent the last phase of posture in this particular pace. *A*, second phase of the support element; *B*, first phase of the flexion element; *C*, second phase of the flexion element; *D*, first phase of the support element.

Compare this statement with the data of Figures 1 and Table 1. This illustrates how misleading and confusing grouping of muscle movements, as such and as they appear in certain reflexes, may become. The following quotation from Mackenzie[6] gives a different and sounder point of view, but one which leads to confusion when applied to Walshe's terms.

> The front of the leg and dorsum of the foot belong morphologically to the general extensor surface of the limb, corresponding with the back of the forearm and the dorsum of the hand. The back of the leg and sole of the foot correspond with the front of the forearm and palm of the hand, so that what we call flexion is really overextension. It is usual, however, and it will be followed here, to use the term flexion for the movement by which the dorsum of the foot is bent toward the front of the leg; the extension for the movement by which the sole of the foot is directed toward the back of the leg.

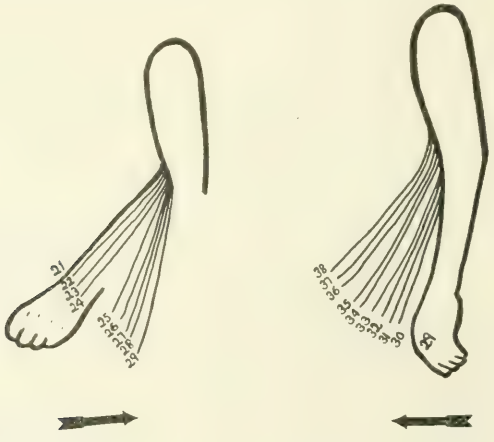

Fig. 11.—This picture illustrates the movements at the elbow as shown by the arrows.

Numerous other similar quotations could be given.

Movements of the Knee.—If a series of tracings of moving pictures is analyzed as far as movements of the knee are concerned, it is found that at the stage when the hip has ceased its backward or ventral movement, the knee is in direct alinement with it (Fig. 13). In the next tracing the hip starts to move forward or dorsally, while the knee starts to move backward or ventrally. This illustrates two points: 1. The ventral position of the leg, as has been previously contended, is one which leaves entirely out of consideration the knee and ankle and controls the leg as a rod. 2. Superimposed on this is the flexion phase which begins with the process of carrying the leg forward—dorsally. When the ventral movements of the hip have ceased, that

6. Mackenzie, W. C.: The Action of Muscles, New York, Paul B. Hoeber, 1918.

is, when the hip starts to move forward, or dorsad, if the movements at the knee are successively traced, it is found that the flexion reflex is at its maximum at a point of time midway between the assumption at the hip of the ventral and dorsal positions, as would be expected (Figs. 9 *B* and 10 *C*). As this point of maximum flexion reflex state is passed, the knee begins to extend until, in the dorsal position, it is again in alinement with the hip, producing a rodlike appearance (Fig. 10 *C*). When the successive stages between the forward (*dorsad*) and the backward (*ventrad*) positions are traced (Figs. 10 *D* and 9 *A*), that is, passing from dorsal through support into ventral, it is found that there is a little flexion at the knee at a point midway between the dorsal and ventral positions and corresponding to the maximum flexion in the other half of the pace. In the sequence ventrad-dorsad it is

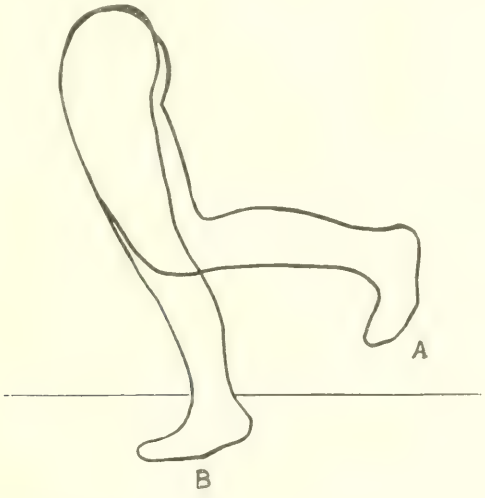

Fig. 12.—This illustrates the change in the location of the posterior thigh line, owing to contraction of the hamstring group of muscles, *A*, and indicates that the anterior thigh line must be used as a constant in comparing successive tracings of the thigh. *A* shows the contour in flexion at the knee; *B*, the contour in extension at the knee.

also found that the hip of the leg of support is directly opposite the hip of the leg of progression (Fig. 5). Thus it appears that:

1. The movement at the hip is diphasic and passes from the ventral to the dorsal position with almost pendulum-like regularity (Figs. 7 and 8).

2. The movements of the knee are tetraphasic. Two knee phases occur during each hip phase (Figs. 9 and 10).

3. The knee phases occurring during the passage of the hip from ventral to dorsal (Figs. 9 *B* and 10 *C*) showed an excursion about twice as great as those occurring during the passage of the hip from dorsal to ventral (Figs. 10 *D* and 9 *A*).

4. On account of its anatomic make-up, though the actual movements carry the foreleg dorsad, the knee never goes into a truly dorsal position.

Flexion of the Knee in the Position of Support.—Furthermore, an unexpected finding has been noted, namely, that the knee is slightly flexed at the phase of maximum support (Figs. 5, 9 *A* and 10 *D*), corresponding to the flexion phase in the other leg and to a phase midway between the dorsal and ventral hip positions. If the particular tracing in which this is found is examined, it is noted that there is forward tilting of the torso, that is, that the center of gravity is not the same as in standing but is thrown slightly forward (Figs. 7 and 8). This slight flexion at the knee in the stage of support appears to be

Fig. 13.—This illustrates the ventral position (left leg) and the dorsal position (right leg)—the first two elements of walking. Compare with Figures 5 and 14.

an adaptation to the forward placing of the center of gravity. As expected, in running, when the center of the gravity is still further forward, flexion of the knee in the phase of support was observed to be still greater. The oscillations of the knee into this slight stage of flexion (Fig. 10 *D*) and out of it (Fig. 9 *A*) are very much smaller than those which occur in the flexion phase while passing from the ventral to the dorsal positions (Figs. 9 *B* and 10 *C*).

In the analysis of reflex stepping by Sherrington, previously alluded to, it was stated that in the "extensor" or antigravity phase (support), the knee was extended. In normal standing, this is the case. However, in the process of moving forward the pushing forward of the center

of gravity somewhat negates this standing position. The importance of this in analyzing the condition known as decerebrate rigidity or anti-gravity posture, whether produced experimentally in animals or as a result of disease in man, is considerable; for, whereas the position of the legs in standing represents that reflex which Sherrington calls "the static of erect posture," the positions in walking and running represent the static posture plus a reaction to the forward placing

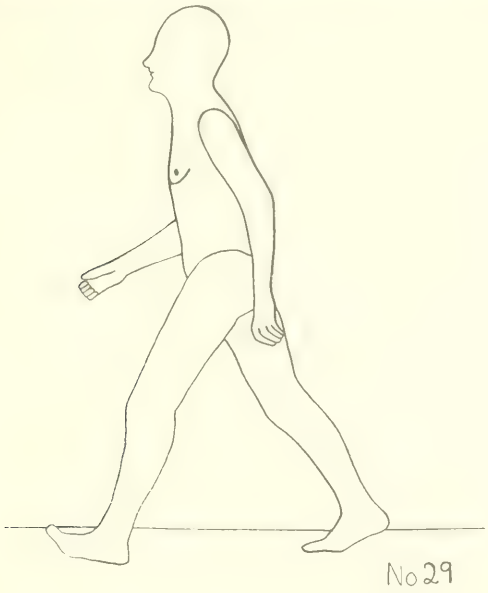

Fig. 14.—This illustrates the ventral position (right leg) and the dorsal position (left leg)—the first two elements of walking. Compare with Figures 5 and 13.

of the center of gravity. The importance in accounting for the flexion of the knees in the posture of parkinsonian syndromes has been alluded to in another article on that subject.[7] The importance of this point in relating postural defects of hemiplegia, pseudobulbar paralysis and the parkinsonian syndromes will be discussed at length another time. However, here it may be remarked that the unilateral defect of the person with hemiplegia represents involvement of the appendicular muscles only. No change of the torso in relation to the center of gravity occurs since the axial muscles having to do with this are not affected. When, however, bilateral involvement occurs, as in the parkinsonian syndrome and pseudobulbar palsy, the axial muscles are

7. Kraus, Walter M.: An Interpretation of the Posture of Parkinsonian Syndromes in Terms of the Neuromuscular Mechanism, New York State J. M. **22**:9, 1922.

affected and the torso bends forward. As a consequence and as a reaction to the forward placing of the center of gravity, flexion at the knee analogous to that seen in the phase of support in walking appears. That group of neurons or integrations of the nervous system which maintains erect posture is apparently affected. The position of the leg in these three conditions is therefore primarily the same. Secondarily, flexion at the knee occurs in bilateral parkinsonian and pseudo-bulbar syndromes, due to the bilateral involvement.

The position of the arm in the parkinsonian syndrome and in pseudobulbar palsy may easily be shown to be, from the neuromuscular point of view, the same as that of typical contracture in hemiplegia. This may be demonstrated by assuming the position of the arm in the parkinsonian syndrome and then successively contracting all those muscles which have produced this position, namely, the ventral group.

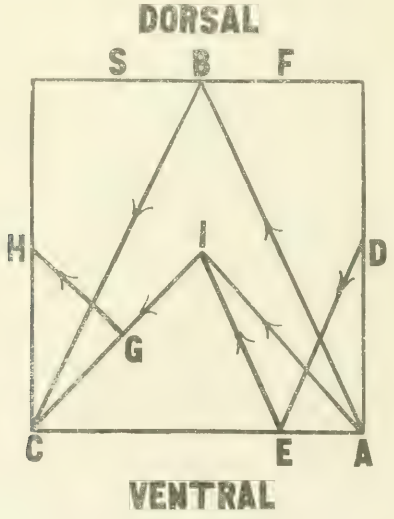

Fig. 15.—Graphic representation of the movements at the hip, shoulder, knee and elbow on one side of the body during a pace of a normal man. *A, B, C,* shoulder and hip. The movement begins at the ventral position, goes to the dorsal position and returns—diphasic; *D, E, I,* flexion phase at the knee—diphasic; *I, G. H,* support phase at the knee—about one-half the extent of *D, E, I*—diphasic. Note that these movements are all from a neutral position midway between ventral and dorsal, into a ventral position and out of a ventral position, back only to a neutral position midway between ventral and dorsal. The knee movement in a complete pace is tetraphasic. *A, I, G, C,* flexion movement at the elbow—diphasic. This shows the same limitations to the ventral field as *D, E, I* and *I, G, H,* the knee movements. The hip and shoulder movements are represented by one line, indicating that though apparently opposite in direction they are really, from a neuromuscular point of view, identical. *F,* maximum flexion; *S,* maximum support.

From this it may be seen that the entire hemiplegic position, from the point of view of posture or attitude, represents half of the position of the parkinsonian syndrome and pseudobulbar palsy, minus the reaction at the knee, which, in these latter diseases, is due to the forward placing of the center of gravity.

Identity of Homolateral Movements of the Arm and Leg.—In the earlier parts of this paper it was indicated that by means of the present method of interpretation of movements by the neuromuscular mechanism, it can be shown that the homolateral movement of the arm and leg represents the activity of the corresponding groups of muscles. Reference to the table will show that the leg is carried backward at the hip by a ventral group of muscles. Duchenne[8] has shown that the gluteus maximus does not participate in either walking on the level or standing. This old observation, which I have been able to confirm, forms a final proof that the backward movement or extension at the hip in walking, is entirely ventral.

The forward movement of the arm is carried out by the pectoral muscles, also a ventral group. Thus it is seen that when the leg is carried backward and the arm carried forward both movements are brought about by ventral muscles. When, on the other hand, the leg is carried forward and the arm backward, both movements are carried out by dorsal muscles (Figs. 13 and 14).

At the elbow there is a ventral and a dorsal swing which coincides with the ventral and dorsal swing at the shoulder. The movements are diphasic (Fig. 11) unlike those of the knee, which are tetraphasic (Figs. 9 and 10). The shoulder movements are diphasic like those at the hip.

Figure 15 represents the most important observations of this article in graphic form.

V. SUMMARY

Analysis of the phases of normal human walking, in the light of knowledge of the embryology of muscles, their grouping and nerve supply, has shown that the customary interpretation based on ordinary observation of muscle actions is, in many respects, incomplete and incorrect.

The homolateral arm and leg, though apparently moving in opposite directions, are moved by the same group of muscles, ventral or dorsal. From the point of view of control by the nervous system, these apparently opposite movements are identical.

The phases of gait are four: (1) the ventral finlike movement of the lower limbs at the hip, (2) the dorsal finlike movement of the lower limbs at the hip, (3) the flexion reflex element, and (4) the support element.

8. Duchenne, G. B.: Physiologie des Mouvements, Paris, 1867.

The sequence of these is rhythmic. The order of sequence is 1, 3, 2, 4—1, 3, 2, 4, etc. The first two (1 and 2) represent activity of singly hinged appendages. The second two (3 and 4) represent activity of three-hinged appendages. The first two and the second two each constitute a pair of corresponding opposites. The two elements of the second pair correspond to the flexor (3) and the extensor phases (4) of reflex stepping as described by Sherrington.

In the first two the leg acts like a rod, leaving out of consideration the two large joints at the knee and ankle. The muscular activity of the ventral phase is obviously antagonistic and opposite to that of the dorsal. The alternating muscular activity at the three great joints of the flexion reflex phase is also opposite to that of the support phase.

Analysis of the hip movements shows them to be diphasic. The shoulder and elbow show the same diphasic activity as the hip. Analysis of the knee movements shows them to be tetraphasic. Two phases occur during each hip phase. The two hip phases are from the ventral to the dorsal position and from the dorsal to the ventral position. The two knee phases constituting the flexion reflex element of a pace occur in the ventral to dorsal hip phase. The two knee phases constituting the support element of a pace occur in the dorsal to ventral hip phase.

In the flexion reflex phase the knee passes through an arc about twice as great as in the support phase. At the maximum support stage the knee is slightly flexed. This is due to the forward placing of the center of gravity of the body.

In the typical postural defect of hemiplegia only the appendicular muscles on one side are involved. In the typical bilateral postural defect of parkinsonian syndromes and pseudobulbar palsy the appendicular muscles are involved on both sides, as well as the axial muscles on both sides. The facts given in regard to the forward placing of the center of gravity show that the typical postural defect of hemiplegia represents half of the typical bilateral postural defect of the parkinsonian syndrome and of pseudobulbar palsy, minus the reflex to the forward placing of the center of gravity.

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ON THE DEFORMITY OF THE FOOT IN DYSTONIA MUSCULORUM *

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The comparative rarity of flexor rigidity of the lower extremities in cases of epidemic encephalitis would alone justify a report of two cases showing this deformity. However, our main object is to explain the mechanism of production of the foot deformity of the syndrome of dystonia musculorum, (1) by various distinct muscle groups, and (2) by the two very different processes of contraction and of contracture separated and differentiated one from the other by means of ether anesthesia. Hitherto these deformities have not been decipherable in terms of any stereotyped postures. We shall endeavor to show that this may easily be done.

ONSET AND DEVELOPMENT OF EXTENSOR RIGIDITY OF THE LOWER EXTREMITIES

Both cases were instances of epidemic encephalitis in young girls eventually showing the clinical picture of dystonia musculorum deformans.

CASE 1.—R. E., aged 15, complained of a cold, bronchitis and pain in the chest in October, 1919. These symptoms were followed during the subsequent year by continuation of the pain, drowsiness, diplopia, headache, drooping of the right eyelid and "dragging" of the left foot. On Christmas day, 1920, choreiform movements began in both legs, which prevented the patient from sitting still. Two days later these movements began in the upper extremities, and two days after that in the head and neck. She was admitted to Bellevue Hospital in January, 1920, where she remained five months. There the choreiform movements continuously carried the trunk and extremities into the posture originally described by Sherrington in experimental decerebration at upper pontile levels. She was admitted to Montefiore Hospital Sept. 29, 1921, showing the picture just described.

CASE 2.—J. S., aged 17, complained of tiredness and sleepiness shortly after January, 1920, that is, about the same time as R. E. She could not sleep well. In August she had fever and pains in her legs. In January, 1921, that is about the same time as R. E., movements of the hands, feet and head began, following

* Read at the Forty-Ninth Annual Meeting of the American Neurological Association, Boston, June 2, 1923.

Fig. 1 (Case 1).—At this stage R. E. showed little permanent deformity. Here she was still able to stand assisted.

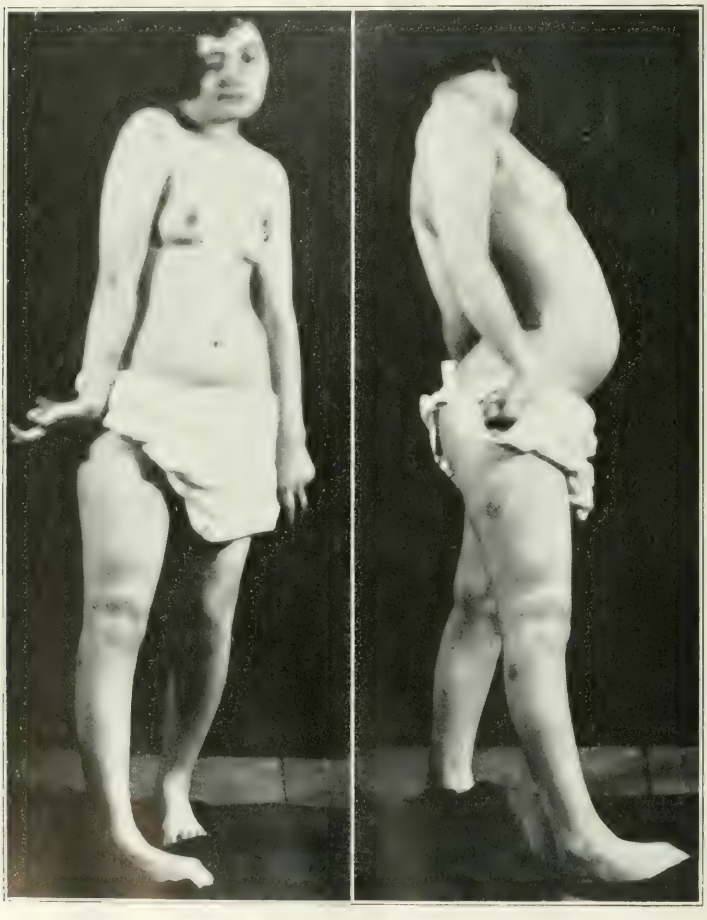

Fig. 2 (Case 2).—The patient is still able to stand alone. Note the ventral foot and extended legs, particularly the right.

a family quarrel and a severe emotional disturbance. Two days later fever and diplopia were present. In May she had an attack of vomiting and abdominal pain. Her pain persisted on and off until March, 1922, when she was admitted to Mt. Sinai Hospital complaining of abdominal pain and mental depression. She became noisy and excited, and occasionally confused at night. She finally left the hospital, but was admitted to Montefiore Hospital, May 13, 1922.

Here examination showed choreiform movements which were like those of R. E., except that at times the muscles involved were not exclusively those active in producing, in the legs, the extensor type of decerebrate posture.[1] This, as the later development of the case showed, was the beginning of the transition to a flexed condition of the legs.

Fig. 3 (Case 1).—These and subsequent photographs are reproductions from a cinematographic film. R. E. in 1921. These show the foot carried by contractions into the posture subsequently maintained by contracture.

In both patients, the movements produced extension of the lower extremities. In both a mild, permanent equinovarus position of the foot with flexion of the toes was present (Figs. 1 and 2). The move-

1. A detailed account of the muscles involved will be found in a paper by W. M. Kraus and A. M. Rabiner, On the Production of Neuromuscular Patterns by Release of Spinal Integrations After Decerebration, J. Neurol. & Psychopath. **3**:209, 1922.

ments accentuated this. There was moderate rigidity in both lower extremities. The arm conditions will not be considered at all in this paper since they are not pertinent to this discussion.

THE TRANSITION BETWEEN THE MOBILE AND IMMOBILE PRODUCTION OF THE EXTENSOR POSITION. "THE VENTRAL FOOT"

Moving pictures of R. E. (Fig. 3) taken in February, 1922, show that the movements carried the foot from a position of rest into that just described. The same occurred in J. S. Gradually, however, the feet became rigidly held in that position, though not equally on the two sides. Since the muscles producing that deformity all developed on the ventral aspect of the limb and were all supplied by ventral nerves, we have substituted the phrase "ventral foot" for a lengthy description of movements such as extension at the ankle, adduction at the ankle and flexion of the sole and toes. This phrase indicates very briefly not only the anatomic origin of the causative nerves and muscles, but emphasizes the fact that they constitute the distal part of a definite embryologic group.

THE CHANGE FROM EXTENSOR TO FLEXOR RIGIDITY

During the late summer and fall of 1922, the lower extremities gradually assumed a flexed position. Some choreiform movements in this new direction persisted for some time, but soon disappeared at all joints, except occasionally at the hip, due to their prevention by increasing rigidity. With the exception of the ankle and foot, the *posture* was that seen in paraplegia in flexion (Figs. 4 and 5). We cannot speak of these cases as "para*plegia* in flexion" since there was *no loss of the ability to initiate voluntary power but only almost complete loss of its manifestations due to rigidity*. The suffix "plegia" we feel should be limited to conditions of flaccid paralysis following loss of voluntary power.

The feet, as the photographs show (Figs. 4 and 5) were characteristic of dystonia musculorum deformans, but in no way characteristic of simple "paraplegia in flexion," for in this condition there is no pes cavus, no flexion of the toes, not very much adduction and no extension of the foot (its relation to the anterior surface of the leg is one of an angle of 90, rather than one of 180 degrees as in extensor rigidity, "paraplegia in extension").

The sequence of extensor rigidity followed by flexor rigidity led us to suppose that there remained a residuum of the postural defects of the extensor phase *caused by contracture* which complicated the picture. In brief, the ventral foot persisted in a posture in which contractions

of dorsal muscles control the posture of the foot. The accompanying table shows the leg muscles divided into dorsal and ventral groups. It was our presumption that the muscles of Column B, Group III, remained set by contracture, while the muscles in Column A were being activated

Fig. 4.—*A*, R. E. (Case 1), note the extreme flexor rigidity and the foot deformities. *B* (Case 2), note the extreme rigidity causing the legs to be held in flexion even when the patient is suspended. Note the foot deformities.

by contractions. Were these muscles alone determining its posture the deformity of simple paraplegia in flexion would have been present. It

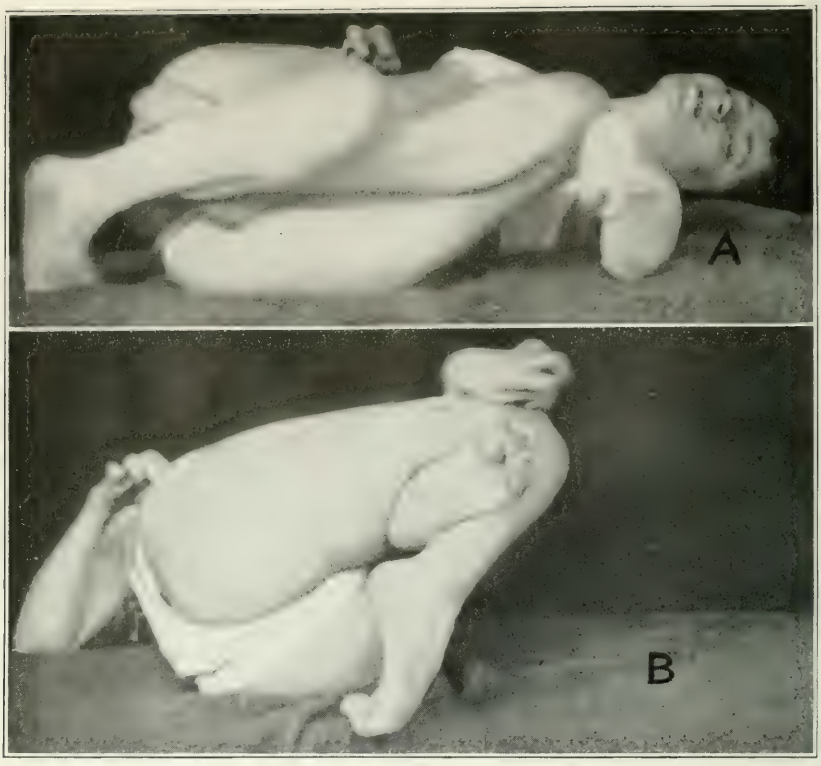

Fig. 5 (Case 2).—*A,* note the extreme flexor rigidity particularly of the left foot and the deformity of the right foot; *B,* note, in addition to the flexor rigidity, the deformities of the right foot, often called the "semilunar foot."

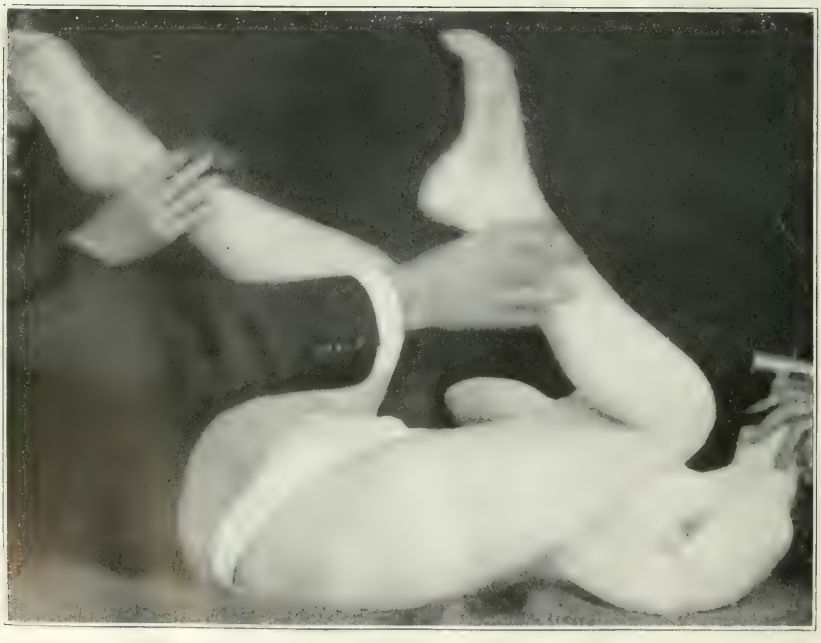

Fig. 6 (Case 2).—Note the flaccidity under anesthesia. The legs are being supported to demonstrate the remaining contractures of the foot muscles. Note also the "semilunar foot" on the left.

was determined therefore to try to separate contracting from contractured muscles by means of ether anesthesia. Figures 6 and 7 show the extreme flaccidity associated with residual flexion of the toes and pes cavus. These positions were quite different from those present when the patients were not under ether. These contractures could only be partially overcome by very forceful traction, while those of the sole and toes could not be overcome at all.

THE RÔLES OF CONTRACTION AND CONTRACTURE

By study of the accompanying table and the photographs it becomes apparent that the contractures of the muscles producing the "ventral foot" quite spoiled the simple picture of the "dorsal foot" of "paraplegia

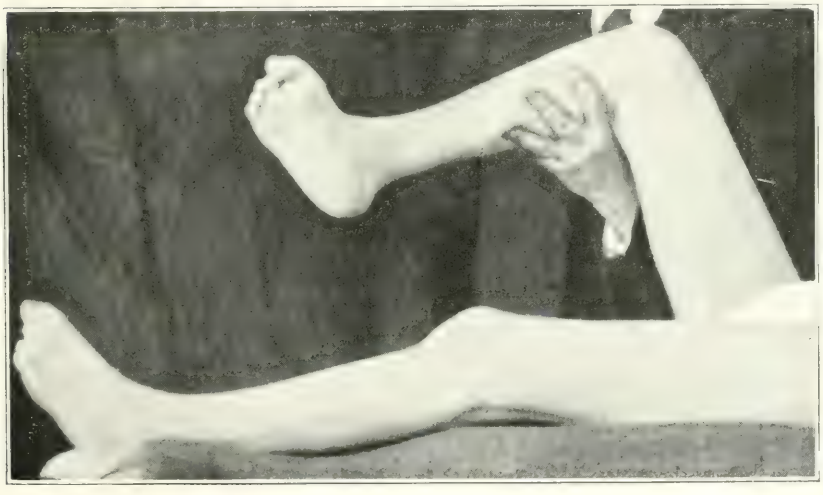

Fig. 7 (Case 2).—Note the flaccidity under anesthesia. The flexion posture is gone, but contracture maintains, pes cavus, flexion of the toes and inversion at the ankle.

in flexion" in which the extensors of the toes are active as well as the peronei and tibialis anticus, which latter balance each other's lateral action (see table). A combined "dorsoventral foot" resulted, whose position as regards inversion was due to the combined action of the tibialis anticus and tibialis posticus. The turning of the sole so that it lay in the longitudinal plane of the leg was facilitated by the position of the foot. It was held by the contractured gastrocnemius on the one hand and the contracting flexor muscles of the ankle on the other, in a position at a little more than a right angle to the leg.

This explanation of the mechanism of production of the foot deformities in the dystonic forms of epidemic encephalitis due to com-

bination of contracture and contraction may be applied to other forms of dystonia musculorum. It is quite apparent that transitional and incomplete varieties of this dorsoventral foot may occur, dependent on the intensity of the contractures and the degree to which the various postural elements composing the classical picture such as flexion, extension, adduction and so forth, at various joints of the ankle and the foot are present. These incomplete and transitional forms must depend on correspondingly incomplete and patchy lesions of the central nervous system.

*The Muscles Active in Flexor Rigidity (A) and in Extensor Rigidity (B)**

Group	A		B	
	Ventral	Dorsal	Ventral	Dorsal
I	†Pectineus (Fl.& Ad. hip)	Iliopsoas (Fl. hip) Tensor fasciae femoris (Fl. hip) Sartorius (Fl. hip) †Pectineus (Fl.& Ad. hip)	Adductors (Ad. hip) Semimembranosus (Ext. hip) Semitendinosus (Ext. hip) Biceps femoris (Ext. hip)	
II	Gracilis (Fl. & Ad. knee) Semitendinosus (Fl. knee) Semimembranosus (Fl. knee) Biceps femoris (Fl. knee)	.....	.....	Quadriceps femoris (Ext. knee)
III	.....	Tibialis anticus (Fl. & Ad. ankle) Extensor digitorum longus (Fl. ankle, Ext. toes) Extensor longus hallucis (Fl. ankle, Ext. big toe) Peroneus longus (Ext. Ev. ankle) Peroneus brevis (Ext. Ev. ankle)	Gastronemius (Ext. ankle) Soleus (Ext. ankle) Tibialis posticus (Ext. ankle) Flexor digitorum longus } Fl. toes Flexor hallucis longus... } & Ext. Quadratus plantae..... } ankle Abductor digiti quinti ○ Flexor digiti quinti brevis ○ Opponens digiti quinti Interosseus plantaris 1} Interosseus plantaris 2}○○ Interosseus plantaris 3} Interosseus dorsalis 1} Interosseus dorsalis 2} Interosseus dorsalis 3}○○ Interosseus dorsalis 4} Adductor hallucis ○ Lumbricalis 1} Lumbricalis 2} Lumbricalis 3}○○ Lumbricalis 4} Abductor hallucis ○ Flexor digitorum brevis ○ Flexor hallucis brevis ○	

* This does not account for all the muscles of the lower extremities, since observation or testing of all of them has not been possible. In both A and B, Group I includes muscles acting at the hip, Group II those acting at the knee, Group III those acting at the ankle, foot and toes. Fl., flexion; Ext., extension; Ad., adduction; Ev., eversion; ○, action indicated by the name; ○○, extend toes at interphalangeal joints and flex at metacarpophalangeal joints. Dorsal interossei abduct toes, ventral interossei adduct toes.

† Double muscle. Supplied by both dorsal and ventral nerves.

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THE PATHOLOGY OF MOTOR PARALYSIS BY LEAD*

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NEW YORK

I. Introduction.
II. The Term "Neuronitis."
III. The Clinical Aspect of Lead Neuritis.
IV. Review of the Literature Dealing with Pathology.
1. Pathology of the Muscles.
2. Pathology of the Nerves.
3. Pathology of the Roots.
4. Pathology of the Spinal Cord.
V. Conclusions.

I. INTRODUCTION

Discussion of the site of attack, whether peripheral, central or both, in diseases of the peripheral neurons, is still active. A recent reference[1] showing the present attitude in England toward the whole problem of multiple neuritis follows:

> Peripheral neuritis, multiple neuritis and polyneuritis are all terms applied to a complex of symptoms due to disease affecting the peripheral motor and sensory neurons. The disease as a whole is characterized by the facts that many nerves are affected at the same time or in rapid succession, that this affection of the nerve is always bilateral and usually symmetrical and that the more distal parts of the neurons are especially attacked, producing symptoms largely limited to the distal parts of the limbs. *It is important, however, to realize that the conception of the disease as one limited to the peripheral nerves is erroneous* [italics ours]. As will be pointed out, the action of the infection or intoxication falls, in many cases at all events, on the central as well as the peripheral nervous system.

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* Read at the meeting of the Section of Neurology and Psychiatry of the New York Academy of Medicine, April 10, 1923.

1. Feiling: Multiple Neuritis, in Oxford Loose Leaf System of Medicine **6**:647, 1921

Barnes,[2] in 1902, described as "toxic degeneration of the lower motor neurone" several cases which, in certain respects, conformed clinically to the condition called multiple neuritis. Holmes,[3] in 1917, and Bashford, Bradford and Wilson,[4] in 1918, established that in a certain type of infectious polyneuritis the anterior horn cells were involved early and that the changes in the nerve trunks were patchy and both parenchymatous and exudative in type. Walshe,[5] in 1918, demonstrated that in certain cases of localized paralysis which were associated with cutaneous diphtheritic lesions, the infection was carried through the lymphatic channels along the nerve trunk and that symptoms appeared when the infection reached the anterior horn cells. In these cases the nerves themselves were free from pathologic changes. In the widespread forms of diphtheritic paralysis in which the toxins are generalized, the nervous system is involved both centrally and peripherally—that is, both the anterior horn cells and the peripheral nerve trunks are affected. These writers have fairly well established that the pathologic process, in some forms of so-called multiple neuritis, involves the entire lower neuron.

Oppenheim,[6] in his textbook (edition of 1911), suggests that in arsenical paralysis the lesions are both central and peripheral, and thus it would be fair to interpret his statements as meaning that the entire lower neuron is involved in this toxic form of multiple neuritis. Kraus,[7] in a review of the literature of diabetic neuritis, found that: "Satisfactory clinical evidence of primary extramedullary involvement of the peripheral neurons, that is, peripheral neuritis, has not been obtained, either from a review of the clinical and pathologic reports in the literature or from my own experience."

II. THE TERM "NEURONITIS"

To combat the doctrine that the condition called "multiple neuritis" is a peripheral neuritis, is no easy task. Not only must the past literature be reviewed, but experimental, clinical and pathologic studies must be made anew. And, even when these are accomplished, one

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2. Barnes: Toxic Degeneration of the Lower Motor Neurone Simulating Peripheral Neuritis, Brain **25**:479, 1902.

3. Holmes, Gordon: Acute Febrile Polyneuritis, Brit. M. J. **2**:37, 1917.

4. Bradford, Bashford, and Wilson: Acute Infective Polyneuritis, Brit. Quart. J. M. **12**:88, 1918.

5. Walshe, F. M. R.: On the Pathogenesis of Diphtheritic Paralysis, Brit. Quart. J. M. **11**:191, 1918; Post Diphtheritic Paralysis, Lancet **2**:232 (Aug. 24) 1918.

6. Oppenheim, H.: Textbook of Nervous Diseases, Otto Schulze & Co., Edinburgh: 1911, Vol. 1, p. 530.

7. Kraus, W. M.: Involvement of the Peripheral Neurons in Diabetes Mellitus, Arch. Neurol. & Psychiat. **7**:208 (Feb.) 1922.

still has to face the inertia due to the ingrained concept that "multiple neuritis" is a peripheral neuritis—that the lesions are primarily or exclusively (or both) in the peripheral twigs of the nerves.

A satisfactory title for this involvement of peripheral neurons has interested several writers. The term neuronitis, so far as we have been able to find, was first used in this country by Charles K. Mills,[8] in 1898, when he wrote: "With regard to multiple neuritis it is no new thing for me to teach the involvement of both nerve and cord in a large number, if not in a majority, of the cases. Long before the theory of the neuron was thought of, I expressed my belief that in many cases of so-called multiple neuritis both periphery and spinal centers were implicated; in other words, as then expressed, we had a concurrence of multiple neuritis and of generalized poliomyelitis in the same case. . . . These diseases are, therefore, in some cases at least, neither instances of neuritis in a strict sense or of poliomyelitis. They are, perhaps, what might be termed 'neuronitis,' and this term has been suggested, but seems to have an unnatural sound even to a neurological ear."

In 1907, E. Farquhar Buzzard,[9] when speaking of acute toxic polyneuritis, said: "The question of title again presents itself, and there is a general feeling that this group might well have a distinctive name, if only a suitable one could be found. 'Toxic degeneration of the lower motor neuron' is too long, and 'motor neuronitis' is open to popular, if not medicolegal, misinterpretation." In 1919, Foster Kennedy,[10] in describing certain cases forming a subgroup of epidemic encephalitis, labeled them "infective neuronitis." The term "neuronitis" is, therefore, not new, and, though possibly not generally used, has at least come to have a sound to the "neurological ear" which can no longer be designated as "unnatural."

After all, what is desirable is that clinical pictures be described and labeled precisely so as to indicate, as far as possible, their cause, pathology and course. We do not wish to give the impression that we maintain that all conditions now spoken of as neuritis, are neuronitis. But three forms of so-called multiple neuritis—due to certain infections and toxins—are affections of the entire lower motor neuron. In diabetes the intramedullary portions of neurons are probably the primary site of attack.[7]

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8. Mills, C. K.: The Reclassification of Some Organic Nervous Diseases on the Basis of the Neuron, J. A. M. A. **31**:11 (July 2) 1898.

9. Buzzard, E. F.: On Certain Acute Infective or Toxic Conditions of the Nervous System, Brain **30**:85, 1907.

10. Kennedy, Foster: Infective Neuronitis, Arch. Neurol. & Psychiat. **2**:621 (Dec.) 1919.

This critical review aims to show that the same is true in lead paralysis, but refers only to that portion of the literature that gives facts of pathology. No mention is made of references dealing with the subject from the clinical standpoint or in which expressions of opinion as to pathology are based only on clinical evidence.

III. THE CLINICAL ASPECT OF LEAD NEURITIS

Lead paralysis may be acute or chronic, and may appear without preceding evidence of lead poisoning. It usually develops a few weeks after such prodromata as colic and pains in the joints and muscles. Once developed, the involvement may seem to be limited entirely to the lower motor neuron. In adults, the paralysis is commonly found only in the upper extremities; in children, the legs are usually first affected. The paralysis is not always bilateral, much less symmetrical.

In the upper extremities there are three principal types of paralysis: upper arm, lower arm, and Aran-Duchenne type. In the last there is extensive atrophy of the intrinsic muscles of the hand, often without commensurate weakness. As a rule, lead paralysis affects the lower arm muscles supplied by the seventh and eighth cervical, and the first thoracic segments. The preference for muscles supplied by segments of the cord rather than for those supplied by a particular nerve or nerves, gives rise to the query—Why is lead paralysis called a peripheral neuritis? The supinator longus is supplied by fibers from the sixth cervical segment. If, in a musculospiral paralysis, the supinator escapes and the sixth anterior root is normal, there is reason for suspecting that the primary change is in the cord. It would be remarkable for the process to begin in the distal parts of a nerve (in this case the musculospiral), extend upward to the cord and yet not involve all the roots of the nerve. Several writers point out that when the supinator longus is involved in lead paralysis, the biceps and deltoid, which are supplied by the fifth and sixth cervical segments, are also affected. This fact leads to the conclusion that the origin is not peripheral. In addition, there are now a number of reports of cases with the clinical picture of amyotrophic lateral sclerosis. The central motor neuron is, therefore, affected in some cases. Potain[11] (1887) pointed out that the dorsal interosseous artery alone supplies the muscles usually involved in lead paralysis, and suggested that general vascular changes, due to lead poisoning, would first show themselves in these muscles because their supply of blood is poor. In this way he provides a possible peripheral origin for palsy of muscles supplied by particular spinal segments. The explanation is ingenious, but is scarcely acceptable in view of the large number of nervous system findings.

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11. Potain: Sur un cas de paralysie hystéro-saturnine, Bull. méd. **1**:851, 1887.

The disproportion between atrophy and weakness of the intrinsic muscles of the hand in the Aran-Duchenne type of lead paralysis should not be passed by without comment. In curare poisoning the motor end plates are attacked and there is complete loss of the transmission of voluntary motor impulses. In family periodic paralysis it has been suggested that involvement of the motor end plates is responsible for the clinical picture. In traumatic lesions of the peripheral nerve trunks the weakness and atrophy are more nearly commensurate. In recognized forms of muscular atrophy due to chronic and gradual degeneration of the anterior horns, we usually find that the atrophy is greater than the weakness. These facts certainly suggest that the Aran-Duchenne type of lead palsy represents a poliomyelitic rather than a terminal neuritic process.

IV. A REVIEW OF THE LITERATURE DEALING WITH PATHOLOGY

An examination of the spinal cord, the roots, the mixed nerve trunks, the muscles and the blood vessels that supply each of these parts is necessary in every case of lead paralysis to permit a trustworthy conclusion as to the primary causal pathologic process. The scarcity of such reports in the literature of the subject is striking. Again, it is usual to find no mention of the stains used in preparing the tissues. If the Marchi or Weigert stains bring out pathologic changes, it is safe to assume their existence. However, the converse is by no means true. Not until Nissl's work,[12] in 1892, was there a method of demonstrating finer cellular pathology. Hence, many of the older reports must be disregarded when they deny the existence of alterations in the spinal cord. In the great majority of cases reported, there was no examination of the anterior and posterior spinal roots. This omission is all important.

Nerve trunks may show degeneration of moderate degree, and in a given case the cord may show slight or no alteration. But if the posterior roots are found to be normal and changes are present only in the anterior roots; and if some roots only of the segments that enter into the makeup of a nerve trunk are involved, then we have important evidence that the degeneration is due to altered cord structure. Another failure in many reports is lack of attention to the condition of the blood vessels of the cord and nerves; this is particularly important because lead is a general poison that is especially apt to produce arteriosclerosis. Feiling[1] states: "In some cases, indeed, other systems of the body,

12. Nissl: Ueber experimentelle erzeugte Veränderungen an den Vorderhornzellen des Rückenmarks bei Kaninchen mit Demonstration mikroskopischen Präparate, Allg. Zeitschr. f. Psychiat. **48**:675, 1892; Versammlung des psychiat. Vereins der Rheinprovins in Bonn: Nov. 14, 1891.

especially the heart, are equally affected." Finally, if a patient with lead paralysis is found to have also either syphilis, tuberculosis, diabetes mellitus or chronic alcoholism, examinations of his tissues cannot be relied on for information concerning the pathology of this disease.

1. PATHOLOGY OF THE MUSCLES

Many authors have found the muscles involved. The common pathological picture is proliferation of connective tissue and atrophy of muscle fibers. Debove and Renaut [13] (1876) raised the question whether section of a nerve would produce the same changes. Friedlander [14] (1879) stated that he believed lead paralysis to be a myopathy. Although later writers [15] have reported cases of lead paralysis resembling progressive muscular atrophy, Harnack [16] (1878), Friedlander [14] (1879), and Kast [17] (1880) were the only authors to maintain that the primary pathologic change occurs in the muscles. More recent investigations indicate that this view is incorrect.

Summary.—There is no adequate evidence that the primary pathologic changes are in the muscles.

2. PATHOLOGY OF THE NERVES

The process is described as a parenchymatous degeneration, beginning usually in the distal parts of the nerve or nerves involved. Goadby and Goodbody [18] (1909), found hemorrhages in the nerves and muscles of poisoned rabbits, but no changes in the cord. Moreover, they found no degeneration of the nerves. We cannot interpret these findings because they are unique; the experiments need to be repeated. Stieglitz [19] (1892), working on dogs, found the nerves often less affected than the cord. Other writers held that in man the degeneration of nerves is usually more intense than that of the cord. Dejerine [20]

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13. Debove and Renaut: Note sur les lésions des faisceaux primitifs des muscles volontaires dans l'atrophie musculaire progressive et dans la paralysie saturnine, Gaz. méd. de Paris, Series 4 **5**:114, 1876.

14. Friedlander: Anatomische Untersuchung eines Falles von Bleilähmung nebst Begründung der myopathischen Natur dieser Affektion, Virchow's Arch. **75**:24, 1879.

15. Grinker, Julius: Multiple Neuritis Simulating Progressive Muscular Atrophy, J. A. M. A. **49**:830 (March 9) 1907.

16. Harnack: Die Wirkungen des Blei auf den thierischen Organismus, Arch. f. exp. Pathol. u. Pharm. **9**:152, 1878.

17. Kast: Notigen zur Bleilähmung, Centralbl. f. Nervenheilk. **3**:137, 1880.

18. Goadby and Goodbody: A Note on the Pathology of Lead Poisoning, Lancet, **2**:988 (Oct. 2) 1909.

19. Stieglitz, L.: Eine experimentelle Untersuchung über Bleivergiftung mit besonderer Berücksichtigung der Veränderungen am Nervensystem, Arch. f. Psychiat. **24**:1, 1892.

20. Dejerine, J.: Recherches sur les lésions du système nerveux dans la paralysie saturnine, Compt. rend. Soc. de biol., Series 7 **1**:11, 1879.

(1879) raised the question whether lead paralysis is due to an ascending or descending degeneration, and quoted Ranvier as denying the possibility of an ascending neuritis. Popow [21] (1883) stated that Lancereaux [22] (1871), Gombault [23] (1873), Bernhardt [24] (1874), Minor [25] (1877), Eisenlohr [26] (1879), Mayor [27] (1877), and Westphal [28] (1874) incline to the opinion that the pathology is primarily in the peripheral nerves, while Remak [29] (1875), Erb [30] (1874, 1883), Renaut [31] (1878), and Bernhardt [24, 32] (1874, 1878) on clinical grounds, regarded the lesions as primarily spinal.

These differences of opinion reflect the spirited controversy existing in the early decades of the development of neurology, and also reflect the difference of opinion concerning the site of pathologic changes underlying the clinical condition known as multiple neuritis. In other words, some were inclined to believe the condition neuritis, while others considered it a neuronitis (since involvement of the anterior horn would imply a subsequent involvement of the peripheral motor fibers).

Of the cases cited by Popow [21] (1883), several must be discounted. In the first place, tissue staining at that time was not delicate enough to

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21. Popow, N.: Ueber die Veränderungen im Rückenmark nach Vergiftungen mit Arsen, Blei und Quecksilber, Virchow's Arch. **93**:351, 1883.

22. Lanceraux: Saturnisme chronique avec accès de goutte et arthrites uratiques, Gaz. méd. de Paris, **26**:385, 1871.

23. Gombault: Contribution á l'histoire anatomique de l'atrophie musculaire saturnine, Arch. de physiol. **5**:592, 1873.

24. Bernhardt: Zur Pathologie der Radialisparalyse, Arch. f. Psychiat. **5**:601, 1874.

25. Minor, L. S.: On Lead Paralysis, Voyenno M. J., St. Petersburg, **141**:149, 1887.

26. Eisenlohr: Idiopathische subakute Muskellähmung und Atrophie, Centralb. f. Nervenheilk. **2**:100, 1879; Ueber einige Lähmungsformen spinalen und peripheren Ursprunges, Arch. f. klin. Med. **26**:642, 1880.

27. Mayor: Lésions des nerfs intramusculaires dans un cas de paralysie saturnine, Gaz. méd. No. 12. Compt. rend. Soc. de biol., 1877, Paris, 1879, 6th series, Vol. 4, p. 213.

28. Westphal: Ueber eine Veränderung des N. radialis bei Bleilähmung, Arch. f. Psychiat. **5**:776, 1874.

29. Remak, E.: Zur Pathogenese der Bleilähmung, Arch. f. Psychiat. **6**:1, 1875.

30. Erb, Wilhelm: Ein Fall von Bleilähmung, Arch. f. Psychiat. **5**:445, 1874; Bemerkungen über gewisse Formen der neurotischen Atrophie, Neurol. Centralbl. **2**:481, 1883.

31. Renaut: Remarques anatomiques et cliniques sur deux points particuliers de l'intoxication saturnine chronique, Gaz. méd. de Paris, Series 5 **7**:394, 1878.

32. Bernhardt, M.: Ueber Bleilähmung und Subacute Atrophische Spinallähmung Erwachsener, Berlin Klin. Woch. **18**:273, 1878.

show finer cord changes. Eisenlohr's patient [20] (1879) was tuberculous and a chronic alcoholic. Gombault's [33] (1880) reports were not complete. Bernhardt [32] (1878) found cord changes in the cervical region. Westphal's [25] case (1874) was not completely examined. Popow [21] (1883), after quoting a number of others who believed that the primary pathology was in the peripheral nerves, himself concludes that the pathologic changes are primarily in the cord. Other later writers continued the discussion without contributing any new pathologic facts to support their theories.

In 1911, Anglada [34] reviewed the literature and came to the conclusion that lead paralysis is a peripheral neuritis. Others hold that while the nerves are first involved the cord is attacked later. Zunker [35] (1880) and Wilson [36] (1907) are in this group. The latter reports Marie [37] (1904) as follows: "An exception can be made in some forms of muscular atrophy due to lead poisoning. These forms show clearly defined lesions of the anterior horn cells in the gray matter (Von Monakow, Oeller, Oppenheim), and may be grouped with chronic anterior poliomyelitis. Wilson then states: "It may be taken, then, that the muscular atrophy of lead poisoning is, in part at least, attributable to the action of the toxic agent on the motor cells of the cord."

The majority of recent writers find changes both in nerves and cord and reach one of two conclusions: (1) The paralysis is primarily of cord origin; (2) it may begin either in the nerves or in the cord. Lead, according to some, acts primarily on the nerve tissue; according to others, the lead affects the blood vessels (Vierordt,[38] 1887) which, in turn, by becoming diseased produce changes in the nerve tissue.

Summary.—On the whole, then, the case for a solely peripheral nerve origin of lead paralysis is weak. The term neuritis tells but a part of the story.

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33. Gombault: Note sur l'état des nerfs périphériques dans l'empoisonnement lent par le plomb chez le cochon d'Inde, Prog. méd. **8**:181, 1880; Contribution à l'étude anatomique de la névrite parenchymateuse subaiguë et chronique, Arch. d. neurol. **1**:11 and 177, 1880 and 1881.

34. Anglada, J.: Paralysie saturnine generalisée probablement polynévritique, Soc. des sc. méd. Montpelier méd., **32**:427 (April 30) 1911; abstracted in Rev. neurol. **23**:264, 1912.

35. Zunker, E.: Zur Pathologie der Bleilähmung, Zeitschr. f. klin. Med. **1**:496, 1880.

36. Wilson, S. A. K.: The Amyotrophy of Chronic Lead Poisoning: Amyotrophic Lateral Sclerosis of Toxic Origin, Rev. Neurol. & Psychiat. **5**:441, 1907.

37. Marie, Pierre: Affections medullaires par intoxications, Traité de méd.—Masson et Cie, Ed. 2, Paris: 1904, **9**:599.

38. Vierordt: Zur Frage vom Wesen der Bleilähmung, Arch. f. Psychiat. **18**:48, 1887.

3. PATHOLOGY OF THE ROOTS

Fisher[39] (1892), Laslett and Warrington[40] (1898), Philippe and Gothard[41] (1903), found changes in the anterior roots only. Stieglitz[19] (1892) and Spiller[42] (1903) found the posterior roots and ganglions also affected. Popow[21] (1883) and Spiller and Longcope[43] (1906), found the roots normal. De Watteville[44] (1880) concluded that the primary lesion is in either the cord or the roots. Ceni[45] (1897) found changes in the cord, roots and muscles. These are the only writers who describe root changes, accompanied as well by changes in the nerves and cord. The presence of root changes by no means indicates that the origin of lead paralysis is in the roots. But such findings are of great importance in determining whether the primary change is in the nerves or the cord.

Summary.—Insufficient attention has been paid to the condition of the roots to warrant definite conclusions.

4. PATHOLOGY OF THE SPINAL CORD

Changes may be present in the meninges, the blood vessels, the gray or the white matter of the cord. The gray matter and vessels commonly show some alteration. Bernhardt[24] (1874), Kussmaul and Maier[46] (1872), Fisher[39] (1892), Stieglitz[19] (1892), Nissl[12] (1892), all remark on vascular congestion or the presence of capillary hemorrhages. Schaffer[47] (1893) found changes in the cords of dogs. Thickening of the meninges is described by Bernhardt[24] (1874) and by Fisher[39] (1892). The majority of writers do not comment on these two points. Mosny and Harvier[48] (1908) found meningeal involve-

39. Fisher, E. D.: Lead Poisoning with Special Reference to the Spinal Cord and Peripheral Nerve Lesions, Am. J. Med. Sc. **104**:51, 1892.

40. Laslett and Warrington: The Morbid Anatomy of a Case of Lead Paralysis, Brain **21**:224, 1898.

41. Philippe and Gothard: Contribution à l'étude de l'origine centrale de la paralysie saturnine, Rev. neurol. **11**:117, 1903.

42. Spiller, W. G.: The Pathological Changes in the Nervous System in a Case of Lead Poisoning, J. Med. Res. **5**:142, 1903.

43. Spiller and Longcope: Multiple Motor Neuritis, Including Landry's Paralysis and Lead Palsy, New York Med. Rec. **70**:81 (July 21) 1906.

44. De Watteville: On the Pathogeny of Lead Palsy, Lancet **2**:44 (July 10) 1880.

45. Ceni: Sur la pathologie de la paralysie saturnine, Arch. f. Psychiat. **29**:566, 1897.

46. Kussmaul, A., and Maier, R.: Zur pathologischen Anatomie des chronischen Saturnismus, Deutsch. Arch. f. klin. Med. **9**:285, 1872.

47. Schaffer: Ueber die Veränderungen der Nervenzellen bei experimenteller chronischer Blei Arsen und Antimon-Vergiftungen, Ungar. Arch. f. Med. **2**:43, 1893.

48. Mosny and Harvier: L'amaurose saturnine, Trib. méd. **46**: 1908.

ment with lymphocytes in the cerebrospinal fluid. Degenerative processes in the anterior horns, most intense in the lower cervical region, were found by Bernhardt [24] (1874), Vulpian [49] (1879), Von Monakow [50] (1880), Zunker [35] (1880), Oeller [51] (1883), Oppenheim [52] (1885), Schultze [53] (1885), Oppenheim and Siemerling [54] (1887), Eichorst [55] (1890), Nissl [12] (1892), Stieglitz [19] (1892), Laslett and Warrington [40] (1898), Spiller [42] (1903), Bernard and Salomon [56] (1903), Philippe and Gothard [41] (1903), Spiller and Longcope [43] (1906) and Claude and Loyez [57] (1914). Of these, Zunker (pathologic findings) and Oeller (nephritis case) have been criticised by Spiller. Birdsall [58] (1882) reported a case in which he considered that cord changes were present. But in the discussion that followed, E. C. Spitzka disagreed on that point and Putnam-Jacoby suggested that the increased vascularity of the affected parts might indicate slackened circulation which could lead to impaired nutrition of the nerve tissues. This case must be disregarded on the score of difference of opinion among those who examined the tissues—even if, for the sake of argument, the staining technic was deemed sufficient to show finer pathology. Dreschfeld [59] (1885) reported the cord normal in his only necropsy. Ceni [45] (1897) found changes in the cord, roots and muscles.

Alterations in the white matter of the cord were noted by Fisher [39] (1892), Goldflam [60] (1893) and Eichorst [55] (1890).

Summary.—The commonest pathologic changes in the cord are in the vessels and the gray matter. The degree of involvement of the

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49. Vulpian: Leçons sur les maladies du système nerveux, Paris **2**:158, 1879.

50. Von Monakow: Zur pathologischen Anatomie der Bleilähmung und der saturninen Encephalopathie, Arch. f. Psychiat. **10**:495, 1880.

51. Oeller: Zur pathologischen Anatomie der Bleilähmung, Munich: 1883.

52. Oppenheim, H.: Zur pathologischen Anatomie der Bleilähmung, Arch. f. Psychiat. **16**:476, 1885.

53. Schultze: Ueber Bleilähmung, Arch. f. Psychiat. **16**:791, 1885.

54. Oppenheim and Siemerling: Beiträge zur pathologischen Anatomie der Tabes Dorsalis und der peripherischen Nervenerkrankung, Arch. f. Psychiat. **18**:487, 1887.

55. Eichorst: Beiträge zur Pathologie der Nerven und Muskeln, Virchow's Arch. **120**:217, 1890.

56. Bernard and Salomon: Un cas de paralysie saturnine à type radiculaire, Rev. neurol. **11**:722, 1903.

57. Claude and Loyez: Etude d'un cas d'intoxication saturnine avec paralysie et atrophies musculaires de date ancienne, Encéphale **9**:30 (July) 1914; abstracted in Rev. neurol. **30**:288, 1914.

58. Birdsall: A Contribution to the Pathological Anatomy of Lead Paralysis, Am. J. Neurol. & Psychiat. **1**:176, 1882.

59. Dreschfeld: On Alcoholic Paralysis, Brain **8**:433, 1885.

60. Goldflam: Ein Fall von Bleilähmung, Deutsch. Zeitschr. f. Nervenheilk. **3**:343, 1893.

nerves and cord varies from case to case. Involvement of the white matter may, at times, produce pseudotabes and lateral sclerosis.

It is granted by most recent authors that pathologic changes exist in the spinal cord. Some writers hold that the changes are primary; some maintain that the anterior horn changes are a "réaction à distance"; others believe that in typical and early cases of lead paralysis the cord is not involved; and finally, some (e. g., Vierordt, 1887) suggest that, since lead is a general poison and produces marked arterial changes throughout the body, the site of election for the primary pathology will depend on individual factors in each case. Erb [30] (1883) and Remak [61] (1862) were the first to suggest that the pathology is primary in the cord, regardless of the possible absence of demonstrable cord changes. They considered that functional impairment of the cord produced degenerative processes in the distal parts of the peripheral nerves. Spiller [42] (1903) stated:

> I conclude, therefore, from my study of this case and of the literature that lead affects the brain and its pia, the nerve cells of the anterior horns of the spinal cord, the ganglia on the posterior roots, the peripheral nerve fibers, and the muscles.
>
> It seems impossible to determine whether its effects are first manifested in alteration of the peripheral motor fibers or of the motor cell bodies of the spinal cord, but inasmuch as *both peripheral nerve fibers and motor cell bodies are sooner or later affected* [italics our], this question is not a very important one.
>
> The condition is, therefore, essentially a peripheral neuron disease or neuronitis.

V. CONCLUSIONS

In estimating the value of past contributions it seemed justifiable to eliminate from consideration case reports in which conspicuously incomplete pathologic examinations were made. Expressions of opinion based on clinical observations, without pathologic facts, have to some extent been discounted. Reports prior to Nissl's work (1892) that the spinal cord was normal, or in which the stains used are not mentioned, have been viewed with question.

In further investigations we feel that a study of root involvement is important. If it should be found that only a part of those entering into the make-up of a motor nerve trunk are affected, the obvious conclusion is that the primary change is central to the roots. Otherwise it would be necessary to explain how a disease process commencing in the distal parts of a motor nerve could extend centrad and fail to involve all the roots connected with that nerve.

It seems to us that the theory of "réaction à distance" does not account for the changes found in the anterior horn cells. The presence

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61. Remak, E.: Ueber die Heilbarkeit der progressive Muskelatrophie, Oesterr. Zeitschr. f. prakt. Heilk. **2**:35, 1862.

of pleocytosis in the spinal fluid and of pathologic changes in the meninges and white matter of the cord, accompanied by marked vascular congestion, features that have been reported by several authors, strongly favor the hypothesis that changes in the anterior horn cells are primary.[62]

For over fifty years there has been a controversy as to whether the origin of lead paralysis is neural or neuronal—whether the entire lower neuron or only its peripheral parts are affected. The trend of both facts and opinion in the last twenty years is in favor of the concept that the entire neuron is involved from the onset of symptoms.

In view of the changing conception of multiple neuritis consequent on the work of Holmes, Bradford and Bashford, Wilson and Walshe, it seems to us that lead paralysis, hitherto called a toxic form of multiple neuritis, should henceforth be regarded as a neuronitis.

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62. Additional references of interest are:

Buzzard, Thomas: Two cases of Lead Paralysis, Brain **1**:121, 1878.

Moritz, S.: A Contribution to the Pathological Anatomy of Lead Paralysis, J. Anat. & Physiol. **15**:78, 1880.

Möbius: Ueber einige ungewöhnliche Fälle von Bleilähmung, Centralbl. f. Nervenheilk. **9**:6, 1886.

Putnam: Lead Poisoning as a Cause of Muscular Incoordination, Boston M. & S. J. **113**:73 (July 28) 1887.

Dejerine-Klumpke, T.: Des polynévrites en général, des paralysies et atrophies saturnines en particulier, Thése de Paris, 1889.

Jolly: Ueber Blei und Arseniklähmung, Deutsch. med. Wchnschr. **19**:97, 1893.

Gowers, W. R.: Lead Palsy, Clinical Lectures, Series 1, London: 1895, p. 237.

Remak, E.: Neuritis and Polyneuritis, Vienna: 1900.

Villaret: Paralysies saturnines, Gaz. d. hôp. **76**:149 (Feb.) 1903.

Gowers, W. R.: Metallic Poisoning, Clinical Lectures, Series 2, London: 1904, p. 140.

Von Sarbo, A.: Spinale Muskelatrophie in Folge Bleivergiftung, Deutsch. Zeitschr. f. Nervenheilk **19**:249, 1909.

Eichorst: Bleivergiftung und Rückenmarkskrankheiten, Med. Klinik **9**:201 (Feb. 9) 1913.

Oliver, Thomas: Lead Poisoning, New York, Paul B. Hoeber, 1914, p. 147.

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FRACTURE OF THE BASE OF THE SKULL, WITH LOSS OF BRAIN SUBSTANCE, FOLLOWED BY RECOVERY

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NEW YORK

While loss of brain substance is relatively common in fractures of the vault of the skull, its occurrence is quite rare with basal fractures. Rawling[1] observed two cases of such nature. One patient, with escape of brain substance from the right nostril, died a few hours after the injury. The second case was thus described by Rawling:[2]

"A lad, 11 years of age, fell some distance out of a window on to his head. He was admitted under the care of my colleague, Mr. Lockwood. On admission he was unconscious, and was bleeding freely from the right ear and nose. Shortly afterward it was noticed that brain matter was issuing from the right external auditory meatus, sufficient brain matter being obtained to fill a teaspoon. The extensive nature of the brain injury was confirmed by the fact that the left arm and leg were paralyzed for some days. On the fourth day the boy regained consciousness and recognized his relations. From this period onward he made an uninterrupted recovery. I have seen the lad on various occasions, the last time one year after the accident. At that time he was an exceedingly bright and intellectual boy."

Except for this instance, I have found no other case similar to the one here reported, in which the patient recovered.

REPORT OF CASE

L. K., a man, aged 50, admitted to the Second Surgical Division of Bellevue Hospital, Aug. 3, 1921, had been injured a few hours previously in a runaway accident. He was conscious on admission to the hospital, but could give no details of the accident. There was a laceration of the scalp, 2 inches long and extending down to the pericranium, on the left parietal region. Palpation revealed a fissure fracture of the left parietal bone, about 3 inches long. Its direction was diagonally forward and downward, the lowest point being just above the left pinna. Blood and cerebrospinal fluid escaped in quantities from the left external

1. Rawling, L. B.: The Surgery of the Skull and Brain. Oxford Publications, Frowde, 1912, p. 93.

2. Rawling, L. B.: The Surgery of the Skull and Brain, p. 101.

auditory meatus. The canal was filled with brain substance. About 2 drams of brain substance was removed by forceps in cleaning the canal.

The patient was stuporous, but irritable when aroused. His pulse was 60, and full in quality. There was definite weakness of the right side of the face. The right abdominal reflexes were diminished, and the right plantar response was extensor in type. There was no paralysis of the right arm or leg. Astereognosis was present in the right hand. Sensation was normal, except for moderate deafness in the left ear. The patient had a definite sensory aphasia. He misnamed objects, and did not recognize his errors, even when corrected. He could read and write.

The roentgen ray revealed a fissure fracture of the left parietal bone, as found by palpation on admission, and also a fissure fracture of the petrous portion of the left temporal bone.

The patient had a generalized convulsion a few hours after admission, and fundus examination disclosed congestion of the optic disks. As there seemed to be free escape of blood and spinal fluid from the left ear, decompression was deemed inadvisable.

Spinal fluid continued to drain for eight days. The patient was quite irritable, but the aphasia improved rapidly. After twelve days in the hospital, he refused to stay longer. Indeed, it was the patient's opinion that his injury was of little moment and he informed the physicians that, in altercations with his wife, he had often received much harder blows on the head.

A recent examination of the patient (February, 1922) revealed the following: The patient complained of tinnitus in the left ear, vertigo with a tendency to fall backward on assuming the standing position, insomnia and forgetfulness. This forgetfulness was confined to new associations, and particularly to the names of people and places. There were no headaches or constipation. There were still traces of the aphasia, demonstrated in hesitancy in naming objects presented, with occasional errors, corrected after help of the examiner. Physically, there was slight nerve deafness on the left side, and diminution of the right abdominal reflexes

Summary.—(1) Fracture of vault and base, with extravasation of about 2 drams of brain substance through the basal fracture into the left external auditory canal; (2) sensory aphasia, weakness of the right side of the face, and reflex changes evidencing involvement of the right pyramidal tract; (3) recovery sufficient to leave the hospital by the twelfth day; (4) slight focal residual phenomena six months after the injury.

COMMENT

A point of practical importance is worthy of note. The presence of definite signs of localized injury to brain sub-

stance following skull trauma is in itself not adequate reason for an immediate decompression operation.

I owe thanks to Dr. J. A. Hartwell of the Second Surgical Service and to Dr. Foster Kennedy of the Neurological Service of Bellevue Hospital for permission to report this case.

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THE TREATMENT OF RESIDUAL EPIDEMIC ENCEPHALITIS.*

By GEORGE H. HYSLOP. New York.

I. Introduction.
II. Pathology.
III. Physiology.
IV. Symptoms.
Interpretation of Symptoms.
V. The Therapeutic Problem.
Explanation of Table 1.
VI. Summaries and Observations.
VII. Observations on Treatment.
VIII. Conclusions.

I.—INTRODUCTION.

The chronicity of the residual phenomena of epidemic encephalitis is a problem of therapeutic importance. The study here presented deals primarily with the treatment of Parkinsonian residua, which are exceedingly common. Grossman,[1] in the re-examination of 89 cases of epidemic encephalitis, found that 58 per cent showed tremors or irregular involuntary movements; 50 per cent examined a year or more after recovery from the acute stage of the disease had these symptoms. This author estimates that approximately 10 per cent of his cases were becoming progressively worse. In the experience of the Neurological Service of Bellevue Hospital and of the Cornell Dispensary, the great majority of patients complaining of the after-effects of encephalitis present a Parkinsonian picture, with a vast variety of concomitant symptoms. Many of the patients are unable to earn as much as they could before their illness. Some cannot work at all through actual physical impediment. Nearly all have personality changes which often are in proportion to the physical symptoms and suggest that, although the disease does at times impair mentality without any evident physical symptoms, such impairments are due to the patient's reaction to his disability. In any event, these patients justly demand that medicine do what it can to relieve them. Inasmuch as encephalitis and its residua are new to medicine, we cannot make an accurate prognosis in the Parkinsonian cases. It is possible that the pathology of the disease makes complete recovery unlikely in the majority of cases. If so, amelioration of the symptoms is the best that can be done.

* From the Neurological Department, Bellevue Hospital, New York.

The search for effective therapeutic measures compels, as a first step, consideration of the pathology of the disease, the functions of the tissues affected, and the symptoms occurring in the Parkinsonian picture.

II.—PATHOLOGY.

In the acute stages of the disease there occur cellular infiltration about the smaller vessels, varying degrees of degeneration of nerve-cells and fibres, and proliferation of neuroglia-cells. These processes are of greatest intensity in the grey matter of the basal ganglia, mid-brain, and pons. The white matter is involved to a much less extent.

In the chronic stages there may be degenerative changes in the walls of the vessels, some perivascular infiltration (less in the more chronic cases), and pronounced neuroglia proliferation. I have not found reported any extensive observations as to changes in the various nuclei and their nerve-cells. There is scanty evidence of anything like a continued infection in most of these cases.

Inasmuch as in the acute stages the foci of greatest intensity are irregularly scattered, the chronic lesions will have a like distribution. In a given chronic case, for example, there may be lesions in the right basal ganglia, the hypothalamus, and the left posterior longitudinal bundle. These lesions will be where the foci of greatest intensity occurred in the acute stage. It is in this fashion that one can best account for the variety of concomitant symptoms present in a number of cases, all having two or three main symptoms in common.

Until we have studies of serial sections of the entire brain in cases of residual encephalitis, one may only guess at the true nature or extent of the pathological processes, and it will be impossible to understand accurately many of the symptoms, which are of great interest in that they throw light on the functions of the basal ganglia, diencephalon, mesencephalon, and pons.

III.—PHYSIOLOGY.

Various syndromes resulting from definite lesions of the basal ganglia, the diencephalon, and the mesencephalon are known. These structures have as their function the correlation of motor and sensory impulses to and from the cortex. Depending upon the site of a given lesion are the symptoms accompanying it. In addition to a correlative function, these parts of the brain have some influence in metabolism and in the function of the sympathetic nervous system. There are sympathetic cells in the mid-brain, and lesions of the mid-brain and hypothalamus often upset the vegetative nervous system. Leschke[2] points out that alterations in function of the diencephalon, especially of the hypothalamus, may lead to important changes in the vegetative

economy, showing themselves in the form of diabetes insipidus, diabetes mellitus, dystrophia adiposogenitalis, heat regulatory changes, disturbances of pupillary reaction, and abnormalities of sweating and vasomotor activity. He states that in all cases of dystrophia adiposogenitalis lesions are found in the diencephalon, yet are often absent in the pituitary body. This is evidence of the existence of a metabolic centre in the diencephalon. Bing[3] regards the salivation and hyperidrosis of encephalitis as due to diencephalon lesions.

The corpora striata have predominantly a motor function. Lesions here produce tonus and posture disorders, and also in some cases tremors of a certain type. The subthalamic and tectal regions also have a motor function.

The thalamus has a sensory function, and some authorities maintain that its state determines the feeling of well-being.

IV.—SYMPTOMS.

The outstanding symptoms in the Parkinsonian cases are rigidity, tremors of various types, and impairment of associated movements. Concomitant symptoms, which vary in the different cases, are numerous. Novel varieties constantly occur; but certain relatively common ones are worth mention—salivation, hyperidrosis (limited to the face in two cases, to one side of the body in two other instances), exophthalmos (stimulus of the grey commissure of the thalamus produces secretion of tears, dilatation of the pupils, and exophthalmos),[4] strongly positive oculocardiac reflex, disturbance of the respiratory rhythm, and increase in weight. Two cases of diabetes insipidus and one of transient diabetes mellitus were observed. We found no instance of altered function of the sexual organs. Apropos of the salivation, it is sometimes found in paralysis agitans, but in Parkinsonian encephalitis the salivation is intense and quite out of proportion to the other symptoms. The degree of salivation present is thus probably of diagnostic value in a case in which one cannot obtain a history of previous encephalitis.

Interpretation of Symptoms.—The symptoms may therefore be regarded as evidence of disturbance of the static system of motility[5] and also the central ends of the vegetative nervous system.[6] The basal ganglia and the diencephalon seem to be sites of the chief lesions. The nature of the morbid process is still a matter of question. It is still unknown whether the symptoms are due to persistence of the original infection, to a recrudescence, or are merely the result of a destruction of tissue during the acute stage of the disease. Bing holds that a definite pathology is established, and that the remaining syndrome is not due to the presence of the virus.

V.—THE THERAPEUTIC PROBLEM.

Thus, the therapeutic problem in residual encephalitis is one of ameliorating symptoms due to degeneration in, or possibly to active infection of, certain tissues, with attendant impairment of function. General hygiene to maintain the body at its best level of activity is a first requisite. Re-educative measures to offset some of the motor disorders are important. Finally, drugs to counteract symptoms should be sought.

The chief symptoms to be treated by drugs are of two forms—those due to a disturbed static system of motility, and those evidencing a disturbance in the central ends of the vegetative nervous system. Both autonomic and sympathetic divisions are affected in residual encephalitis, and the problem is rendered the more difficult because of the fact that in a given patient the symptoms do not characterize disturbance of either part alone. There is rather a confusion of symptoms, showing that there is an irregular involvement of both divisions. The fluctuations in symptoms often made it difficult to interpret the effects of drugs. Subjective changes and the influence of suggestion had to be considered. All patients but one of a group of fifteen encephalitics, the subjects of this study, were observed while they were on the ward. The period of observation averaged over a month for each case—for some patients as long as three months, and less than a month in only three instances.

Various drugs, including arsenic (both as Fowler solution and in the form of sodium cacodylate), strychnine, iron, urotropine, and the endocrine products were used in these and other post-encephalitics. Physiotherapy—massage, exercises, electrotherapy, and hydrotherapy—was given to nearly all cases. As other observers have learned, these methods of treatment seem to have no real curative effect.

In selecting drugs for experimentation, those having a definite action on the vegetative nervous system were chosen. Central depressants in particular seemed theoretically the most promising. The long-known value of hyoscine, a central depressant, in paralysis agitans, and the points of resemblance between that disease and the Parkinsonian residua of encephalitis, suggested the use of that drug. Other observers have found it helpful in residual encephalitis. Atropine and belladonna, although acting as central stimulants, were used. Belladonna preparations all contain some hyoscine, and therefore their action is difficult to interpret. Gelsemium and its active principle gelseminine are central depressants, and therefore demanded investigation. The action of this drug is not well understood, and it has not been used in residual encephalitis. In addition to these drugs, adrenalin, nicotine, picrotoxin, pilocarpine, and morphine were used, but chiefly as controls and to a less extent. Cicutine hydrobromate

has been reported as of value. It has pharmacologically the same action as coniine, which in turn is similar to gelsemium. For that reason, and also because an opportunity did not present itself, no observations on cicutine were made.

RESIDUAL ENCEPHALITIS CASES.

Case	S.	A.	Interval between acute stage and onset of residual symptoms	Period from acute stage to admission to hospital	Period of observation	Character of residual symptoms prior to admission
1. D. O.	M	19	None	Admitted as acute case	4 months	Stationary
2. P. A.	M	18	None	12 months	3 months	Gradual progression with remissions
3. J. C.	M	30	2 months	5 months	2½ months	Stationary
4. E. E.	M	17	1 month	7 months	1½ months	Gradual progression
5. J. D.	M	24	1 month	12 months	1 month	Gradual progression
6. H. B.	M	32	3 months	18 months	5 weeks	Gradual progression
7. J. W.	M	56	None	16 months	2 months	Stationary 1 year, relapse and progression for 4 months
8. P. B.	F	28	None	14 months	7 weeks	Relapses at 3rd and 9th months, stationary from then on
9. M. S.	F	26	None	20 months	1 month	Stationary
10. E. B.	F	37	None	24 months	6 weeks	Stationary
11. M. L.	M	19	14 months	22 months	6 weeks	Gradual progression
12. J. K.	M	55	2 months	26 months	5 weeks	Stationary, remissions
13. S. F.	F	14	None	18 months	4 months	Relapse 4th month, gradual progression 14 months, with slight remissions
14. P. W.	M	30	None	16 months	3 weeks	Stationary
15. S. J.	M	18	14 months	18 months	1 week	Gradual progression

The major part of the observations were upon the effects of hyoscine and gelsemium. As subjects there were 15 patients with residual symptoms of encephalitis. One patient had choreiform movements; the others were all of Parkinsonian type. One patient was under observation from the time of onset of the disease. The appended

table gives the important details in each case. In addition to the post-encephalities, 5 cases of trigeminal neuralgia were studied for the effects of gelsemium, and 3 paralysis agitans cases were studied for the effects of both gelsemium and hyoscine.

Explanation of Table I.—Of the 15 patients, 4 were female, 11 male. This proportion has held true in our experience of about forty cases of residual encephalitis. In all but two patients the residual symptoms were present from the time of the acute stages of the disease or within a short period after seeming recovery. In 2 instances there was an interval of normal health of fourteen months before new symptoms appeared. In both of these patients the Parkinsonian state developed gradually. Three of the fifteen patients had relapses; one patient (*Case* 7) had a relapse taking the form of bulbar palsy and seemingly had a recrudescence of the infection. In the other two instances (*Cases* 8 and 13) the relapse followed a miscarriage and a fall. In *Case* 3, after two months of nearly normal health, a fright occurred and Parkinsonian symptoms appeared at once. Emotional shock and strain are known frequently to be followed or accompanied by increase of symptoms in various organic diseases of the central nervous system. One may cite brain tumour, multiple sclerosis, cerebral forms of syphilis, paralysis agitans, and cerebral arteriosclerosis. Possibly emotional states produce cerebral circulatory alterations which, in the presence of disease, may further cripple functions already impaired.

VI.—SUMMARIES AND OBSERVATIONS.

Brief case summaries will be given first; observations of particular interest will follow; and finally the salient features of the various cases will be tabulated.

Case 1.—D. O., male, age 19.

History. Onset February, 1921. Parkinsonian type; recovery by April 15. Residua stationary and consisting of slight physical symptoms and marked hypochondriasis. Discharged May 13.

Treatment.—Treatment of residua by hyoscine and gelsemium. No effects. After discharge took gelsemium for four months. No effects.

Case 2.—P. A., male, age 18.

History. Onset January, 1920. Partial recovery by February. Lethargy, weakness, and diplopia until July, when tremor, anteropulsion, and monotonous speech appeared. Gradual progression until admission, January, 1921. Discharged improved in February. Did well about one month, then got gradually worse again. Re-admitted April, discharged June 8.

Treatment.—During first admission improved under arsenic and physiotherapy. During second admission there were several fluctuations

in symptoms, which seemed to be independent of medication. The greatest and most rapid improvement was when hyoscine was being given. Results are quite doubtful therefore.

Case 3. J. C., male, age 30.

History.—Onset December, 1920; recovery in a month. Fright in February, followed at once by tremor, stiff gait, monotonous voice, and stammering. Admitted May 3, discharged July 26, 1921.

Treatment.—Improved about 50 per cent, with occasional brief periods of slight relapse. Opium, given for four days, was accompanied by the most marked improvement. While taking gelsemium improvement was steady.

Case 4. E. E., male, age 17.

History.—Onset November, 1920; recovery for one month. Gradually progressive Parkinsonian symptoms, commencing in January. Admitted July 1, 1921, marked Parkinsonian picture. Discharged Aug. 12.

Treatment.—Gelsemium only. There were slight fluctuations in condition, but in general a tendency toward progression. Drug not effective.

Case 5. J. D., male, age 24.

History.—Onset June, 1920. One month later right-sided rigidity and tremor. August and September, diabetes mellitus. Parkinsonian features gradually progressed. Admitted July 19, discharged Aug. 22, 1921.

Treatment.—Gelsemium only. Steady and rapid improvement, practically well on discharge. Drug effective.

Case 6.—H. B., male, age 32.

History.—Onset January, 1920; recovery in two weeks. April, tremor left side of face, slowness in movement. Gradual increase of Parkinsonian features. Admitted July 29, discharged Sept. 15, 1921.

Treatment.—Improved steadily on gelsemium, got worse during ten days with arsenic, improved again as soon as gelseminine hydrochlorate was given. Discharged with at least 50 per cent improvement.

Case 7.—J. W., male, age 56.

History.—Onset January, 1920, followed by somnolence, rigidity left side, and slowness in all movements. January, 1921, had sore throat, dim vision, salivation, dysarthria, and dysphagia. Progressively worse. Admitted April 5, 1921, discharged June 10, 1921.

Treatment.—Made steady improvement on hyoscine and gelsemium. Relapsed on arsenic; improved again while opium was given; worse under pituitary and adrenal extract. Hyoscine and gelsemium each partly effective.

Case 8.—P. B., female, age 28.

History.—Onset March, 1920, improved until July. Had a miscarriage then, rapidly grew worse with Parkinsonian symptoms, with a severe relapse in January, 1921. Admitted May 20, discharged July 11, 1921. This patient was bedridden and absolutely helpless.

Treatment.—Steady and marked improvement under hyoscine and gelsemium, and under morphine. Made distinctly worse by atropine. In the last two weeks the patient received only urotropine and grew steadily worse.

Case 9. M. S., female, age 26.

History.—Onset March, 1920, followed by slight degree of Parkinsonian symptoms and marked diplopia. Patient very hypochondriacal. Admitted Nov. 7, discharged Dec. 2, 1921.

Treatment. Rigidity slightly decreased by both gelsemium and hyoscine. Adrenalin followed by brief increase in rigidity, nicotine by brief decrease. Other symptoms unchanged.

Case 10.—E. B., female, age 37.

History.—Onset October, 1919, followed by moderate degree of Parkinsonism, possibly a little worse the last six months. Admitted Oct. 16, discharged Nov. 28, 1921.

Treatment.—Improvement with hyoscine, gelsemium, and gelseminine hydrochlorate. All about equally effective. No improvement with opium. Adrenalin and nicotine had same effects as in *Case* 9.

Case 11.—M. L., male, age 19.

History.—Onset December, 1919, with complete recovery in three weeks. Perfectly well until February, 1921. From then on, gradually progressive right-sided Parkinsonism. Admitted Oct. 10, discharged Nov. 28, 1921.

Treatment.—Steady improvement under gelsemium, gelseminine hydrochlorate, and hyoscine, worse when none of these drugs were given. Adrenalin effects as in *Cases* 9 and 10.

Case 12.—J. K., male, age 55.

History.—Onset December, 1918, recovery in one month. In February, 1919, choreiform movement of the left leg, face, and shoulder appeared. This lasted three or four weeks; since then there have been regular recurrences every six to eight weeks, not related to anything that the patient knows about. Admitted April 13, discharged May 11, 1921.

Treatment.—Improvement under gelsemium and hyoscine definite. Reported back to the hospital for three months after his discharge, and stated that as soon as he took gelsemium the movements decreased. Other drugs had no effect.

Case 13.—S. F., female, age 14.

History.—Onset January, 1920. Immediate left-sided Parkinsonian residue, but gradual improvement until August; then fell while skating and at once symptoms increased. After graduating from school in January, 1921, became steadily worse, the rigidity and tremor now being on the right side as well as the left. Admitted Nov. 18, 1921, discharged March, 1922.

Treatment.—Every time hyoscine, gelsemium, or gelseminine hydrochlorate was given there was distinct improvement. Atropine made her worse. Belladonna had no effect. Adrenalin increased rigidity. Picrotoxin, nicotine, and pilocarpine had no effects except for perhaps an hour or two after a dose was given; of these, nicotine decreased rigidity, the others increased it. The patient had four quite definite slumps in her condition, seemingly independent of what medication was being given. About 20 per cent improvement on discharge.

Case 14. P. W., male, age 30.

History. Onset February, 1920, followed by fatigability, tremor, sweating, and slight left-sided Parkinsonism. Stationary. Admitted June 19, discharged July 9, 1921.

Treatment. One week of gelsemium alone made definite objective betterment; addition of glandular substances and strychnine did not alter the rate of improvement. Results here are perhaps open to some question on the score of complex medication. However, the gelsemium started the improvement.

Case 15. S. J., male, age 18.

History.—Onset March, 1920. Perfectly well until June, 1921. Sudden appearance of breathing irregularity, with paroxysms of deep and rapid breathing, accompanied by moderate degree of Parkinsonism. Gradual progression until admission Oct. 18, 1921. Discharged Oct. 24, 1921.

Treatment.—Parkinsonian symptoms immediately improved (some disappearing) under gelsemium. No change in respiratory phenomena.

In addition to the above 15 patients with residual encephalitis, gelsemium and gelseminine hydrochlorate were given to 3 paralysis-agitans and 5 trigeminal-neuralgia patients. Two of the paralysis-agitans patients were helped slightly by these drugs: one, an early case, was improved considerably. Four of the neuralgia patients were helped (one patient remarkably so) in that these drugs gave relief where morphine failed.

In the case summaries, it will be noticed that little mention has been made of the other drugs used, or of the physiotherapy given the patients. That is because of the fact that where favourable results were obtained they seemed to accompany the administration of the drugs hyoscine, gelsemium, or gelseminine hydrochlorate. Again, in residual cases not reported in this paper, where these drugs were not used, little or no relief was obtained. Moreover, space for detailed description of treatment and results would obscure the positive findings in the mass of negative findings.

VII.—OBSERVATIONS ON TREATMENT.

1. Adrenalin 1–1000, in doses of 20 min. subcutaneously, increased the pulse-rate 50 per cent and definitely exaggerated the rigidity of the extremities for a period of two hours in three cases. Similar doses to non-encephalitic control cases had no such marked effects.

2. Nicotine sulphate, $\frac{1}{30}$ gr. subcutaneously, decreased the rigidity for a few minutes in three cases.

3. Picrotoxin, $\frac{1}{16}$ gr. orally three times a day, had no effect in one case. Given subcutaneously to the same patient, there seemed to be a temporary increase in rigidity.

4. Pilocarpine, $\frac{1}{12}$ gr. orally three times a day, had no effect on the one patient to whom it was given.

5. Atropine, $\frac{1}{100}$ gr. five times a day, made the patients worse in two instances (the same effects were noted in one other case not included in this report). Belladonna, as the tincture, in doses varying from 20 to 30 min. given three times a day, had no action, or produced effects similar to those of atropine.

6. Morphine given as opium pills, 1 gr. twice a day, caused improvement during administration in three cases and produced no results in one case.

Of these drugs, those stimulating the central ends of the vegetative nervous system were followed by increase of Parkinsonian symptoms. Action on the peripheral ends of the autonomic seemed to be of slight importance. Morphine, as a central depressant, gave relief; but it is a drug that one could not well give continuously to a sufferer from residual encephalitis.

We now come to the effects of hyoscine, gelsemium, and gelseminine hydrochlorate. Hyoscine was used in doses of $\frac{1}{100}$ gr. three times a day (usually orally). The dose of gelsemium, in the form of fluid extract, was 7 min. three times a day, although in some instances smaller doses were given. Gelseminine hydrochlorate was given in $\frac{1}{30}$-gr. doses three times a day, sometimes by mouth and sometimes subcutaneously; either mode of administration seemed to be equally effective.

Hyoscine was given to eight patients. Six (*Cases* 8, 9, 10, 11, 12, and 13) were definitely helped. Two (*Cases* 1 and 2) were not affected.

Gelsemium or gelseminine hydrochlorate, or both, were given to fifteen patients. Ten were helped (*Cases* 3, 5, 6, 7, 10, 11, 12, 13, 14, 15); of these cases, 5, 6, 11, and 14 responded most noticeably. Two patients (*Cases* 8 and 9) should be classed as doubtful because of the slight and temporary improvement. Three patients were not helped at all (*Cases* 1, 2, and 4). With these drugs, cumulative symptoms, which consisted of heaviness of the eyelids, dim vision or diplopia, dizziness, and at times a feeling of languor or confusion, appeared in about one-third of the patients. When these symptoms appeared, reduction of the dose to 4 or 5 min. thrice daily permitted continuous administration of the drugs. Cumulative effects disappeared within twenty-four hours after withdrawal of the drug, and were alarming in only one instance. That patient was one to whom gelsemium had been given for six days. She then received $\frac{1}{100}$ gr. of hyoscine subcutaneously. Within five minutes of the injection she developed all the symptoms of hyoscine poisoning, and for four hours was absolutely free of Parkinsonian symptoms. This patient had had hyoscine in the same dosage previously without any such effects. So the situation was repeated. The same symptoms recurred. These observations suggest a synergism between hyoscine and gelsemium.

VIII.—CONCLUSIONS.

1. The Parkinsonian symptoms suggest overaction of the central ends of the vegetative nervous system.

2. Drugs depressing the central ends of the vegetative nervous system ameliorate, those stimulating increase, Parkinsonian symptoms.

3. Hyoscine produced objective improvement in 75 per cent of Parkinsonian cases. It reduced tremor and rigidity, restored mobility of facies, and enabled patients to walk and use the arms more freely.

4. Gelsemium and gelseminine hydrochlorate were effective in the same manner as hyoscine in 66 per cent and gave marked relief in 25 per cent of Parkinsonian cases.

5. Hyoscine gives more relief than gelsemium or gelseminine hydrochlorate in most instances where the three drugs are given; but since the latter two drugs have no dangerous side actions, they are preferable to hyoscine for prolonged administration.

6. Two patients seemed to be cured by gelsemium. The degree of relief cannot be predicted from the duration of symptoms prior to treatment, but is inversely proportionate to the severity of the symptoms. The duration of relief corresponds to the period of administration of treatment.

7. In continuing observations on dispensary patients it was occasionally found that while either one of the two drugs (hyoscine or gelsemium) would be of help, the other would be of no avail or might even increase the symptoms. This seldom occurred, and, since continuous and frequent observation of the patients was impossible, no explanation is offered for the seeming contrary action of the drugs.

8. While either hyoscine or gelsemium seems to be indicated in the treatment of Parkinsonian residua of encephalitis, and while these drugs usually proved beneficial as judged by objective improvement during their administration, it must be admitted that the therapeutic problem is not simple. These same drugs at times fail to help. Other drugs have been reported as giving striking relief in certain patients.

One of the most striking therapeutic results was observed by Dr. Kennedy[7] in his private practice: "A woman of middle age who had been unable to articulate, chew, or move the arms or legs on volition—so intense was the Parkinsonian spasm—after a suppository of belladonna was able to talk fluently, move quickly and with grace, and express emotion easily by gesture and facial expression. Congealment of function gradually returned, but temporary dramatic amelioration has always been possible by the rectal use of belladonna in tolerance dosage".

More complete knowledge of the pathology, and of the function of parts affected by the disease, is necessary before we can predict what drugs should prove beneficial in a given instance.

I wish to express my appreciation to Dr. Foster Kennedy, in whose service at Bellevue Hospital these patients were observed, for criticisms and suggestions during the course of this work.

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OBSERVATIONS IN ONE THOUSAND NEUROLOGIC CASES

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My purpose in this paper is to offer a brief statistical analysis and to touch on some of the features of general interest.

The Bellevue neurologic service, which is under the control of the Cornell Medical School, is distinct from the psychiatric service, and has, at present, two wards, each of twelve beds, for men and women patients. There are two interns, a resident in charge, and an assistant. In addition to the charge of their own wards, the interns are on call for consultation work throughout the hospital.

The members of the visiting staff see all patients in the neurologic wards, and most of those examined in consultation in other services. An adequate physiotherapy department is available. The social service department gives its usual aid and, in addition, supplies occupational therapy instructors.

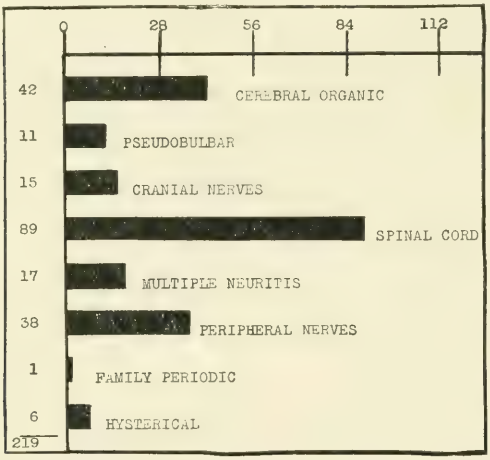
From other wards in the hospital are selected the majority of patients cared for in the neurologic service. During 1921, 300 patients were treated in the ward, and the duration of treatment for each patient averaged twenty-five days. Eight hundred and sixty-two patients were examined in other wards, and many of these were seen several times by the interns. In addition to the hospital patients, several hundred persons were referred from outside the hospital for neurologic examination. These are not included in this report.

There were approximately 43,000 admissions to Bellevue Hospital in 1921, and the neurologic service was not called into consultation in the majority of certain conditions in which neurologic diagnosis could be made without the help of the neurologic service. Only the minority of paralysis agitans, tabes dorsalis, cerebrospinal syphilis, apoplexy, old hemiplegia (the result

of normal apoplexy), epidemic poliomyelitis and hysteria cases were seen by the neurologic service. Two hundred and forty-three fractured skull cases were admitted to the hospital during the year, and the neurologic service was called into consultation in only seventy of these cases.

A small number of psychiatric patients and a similar number of psychoneurotic patients were admitted to the neurologic service.

In the 1,003 patients examined or treated by the neurologic service, 120 different disease conditions were seen. In some patients, the diagnosis included more



Types of paralysis seen in the neurologic service: "cerebral organic" includes tumor, head injury and vascular accident; "spinal cord" includes Friedreich's ataxia and multiple sclerosis.

than one condition—for example, one patient had pseudobulbar palsy on a syphilitic basis; he was admitted to the hospital with a fractured femur, and, as a result of his injury, had an hysterical paralysis of the affected leg. Intercurrent non-neurologic conditions occasionally appeared while a patient was under care in the neurologic ward. For example, one choreic patient developed acute appendicitis. These non-neurologic conditions were not included in the 120 different disease conditions seen.

In Table 1 there are thirteen conditions, each of which was represented by over twenty cases. The

total number of cases in this group is 649, or approximately 65 per cent. of all patients seen during the year.

Eliminating skull injuries, constitutional psychoses and psychoneuroses, constitutional inferiority and mental defects (a total of 216 cases), we have 787 cases of organic nervous disease. Seventeen different conditions were represented by a total of 513 cases, about 66 per cent. of all instances of organic involvement of the nervous system.

TABLE 1.—*Common Conditions in Order of Frequency*

	Seen by the Neurologic Service	Number of Cases Admitted to Bellevue Hospital
Syphilis	117	67*
Fracture of the skull	70	243
Miscellaneous paralyses	69	156
Epilepsy	61	175
Psychoneuroses	59	216†
Psychoses	47	
Epidemic encephalitis	44	
Neuritis of single nerves	38	
Vascular accidents	34	197
Transverse myelitis	33	
Tumors of the brain	31	
Tuberculosis of the central nervous system	24	
Multiple sclerosis	23	
Acute epidemic poliomyelitis	17	
Multiple neuritis	17	
Concussion of the brain	15	
Tumors of the cord	15	
Cerebrospinal meningitis	14	
Constitutional inferiority	14	
Combined sclerosis	13	
Paralysis agitans	12	
General arteriosclerosis	11	
Mental defectives	11	
Brain abscess	10	
Total	798	

* Includes primary, secondary and tertiary, but not tabes or paresis.
† Only hysteria; others not grouped under this heading.

The accompanying chart classifies the anatomic distribution of the various forms of paralysis observed. In family periodic paralysis, the neural mechanism of the paralysis is not known, but the complete blocking of the motor neuron functions suggests a process akin to that seen in curare poisoning, and the analogy may warrant one in suspecting that the motor end plates are involved.

Hysterical paralysis is included in the chart to show its relative rarity, and because a correct diagnosis of hysterical paralysis is of the utmost importance. Several of the six cases seen had previously been regarded

as of organic nature. One exception to this was interesting. A man, aged about 30, was stabbed in the back of the neck. The force of the blow knocked him down. He was unable to get up. The ambulance surgeon found a hemiplegia, and diagnosed it as hysterical. The patient was taken to the hospital, and the admitting staff spent two hours trying to make the patient get up and walk. A careful examination showed a hemisection of the cord at the fifth cervical segment.

Table 3 might itself be the subject for an extensive bit of research, but it is included in this paper without comment because it shows the frequency of definite

TABLE 2.—*Most Frequent Organic Conditions Affecting the Nervous System*

Syphilis	117
Epilepsy	60
Epidemic encephalitis	44
Single neuritis	38
Vascular accident (cerebral)	34
Transverse myelitis	33
Tumors of the brain	31
Tuberculosis of central nervous system	24
Multiple sclerosis	23
Acute epidemic poliomyelitis	17
Multiple neuritis	17
Tumors of the cord	15
Cerebrospinal meningitis, except that of tuberculous origin	14
Combined sclerosis	13
Paralysis agitans	12
General arteriosclerosis	11
Brain abscess	10
Total	513

clinical conditions which, in their association with convulsions, might reasonably be regarded as causally connected. In a large proportion of the patients having generalized seizures, detailed histories showed that early in the course of the disease many attacks were atypical and of the sort described by Gowers.[1]

The sixty cases enumerated in Sections A and B of Table 3 were referred to the neurologic service as "idiopathic epilepsy." After a study of these patients, there were but one third in whom it was impossible to establish either anatomic or physiologic facts which gave us some sort of understanding of the cases. These residual twenty patients, therefore, still had to be regarded as having "idiopathic epilepsy."

1. Gowers: The Borderland of Epilepsy.

In Section C, the seizures were known to be symptomatic at the time of the first examination.

In Table 4, the classifications are in accordance with the Bellevue nomenclature, and do not permit of more detailed analysis of diagnosis or knowledge of etiology or location of the pathologic changes in the central nervous system. Clear psychiatric cases are not included in this table.

During 1921, there were admitted to Bellevue Hospital 38,438 patients with medical conditions. Section 1 of this table enumerates the neurologic conditions.

TABLE 3.—*Epileptic Convulsions*

A. Jacksonian:	
1. With brain tumor	3
2. With tuberculosis of brain	1
3. Posttyphoid	1
4. Arteriosclerotic	1
5. Syphilis	1
B. General:	
1. "Idiopathic"	20
2. With constitutional inferiority or epileptic personality	9
3. Posttraumatic	4
4. Syphilis	3
5. Status epilepticus	3*
6. Hystero-epilepsy	3
7. Due to ear disease	2
8. Sensory epilepsy	2†
9. Pachymeningitis	2
10. Vasovagal attacks	2
11. Static (cerebellar)	1
12. Alcohol	1
13. Gastro-intestinal	1
C. Seizures occurring in thirty-one brain tumors:	
1. Jacksonian	9
2. Generalized	4

* All three cases were "idiopathic" epilepsies.
† Trauma to occiput and osteoma of skull.

The classification is a rather rough one, and it is quite possible that there were included under different headings a great many patients in whom there were neurologic complications. For instance, there were three lobar pneumonia patients who died as the result of metastatic cerebral abscesses. It would be an enormous task to go through all the records to determine the relative proportion of admissions in which there was either an organic or a functional nervous disease, as either a complication of or a concomitant of a medical condition. The enumeration in Section 1 gives a total of 2,851 cases of conditions in which a neurologic examination would be indicated. Of the 673 syphilitic

patients, those in the primary stage would not really need a neurologic examination. There were, roughly, about 150 such cases, but it is difficult to decide where the primary stage ends and the secondary stage begins, and perhaps it is not unjustifiable to maintain that a neurologic examination is desirable for every syphilitic patient. These 2,851 cases represent about 7.4 per cent. of all medical admissions to Bellevue Hospital.

The surgical services admitted about 4,500 traumatic cases in 1921. Fractures of the skull and spine totaled 262, or about 5.8 per cent. of traumatic cases. In order

TABLE 4.—*Neurologic Cases in Bellevue in 1921*

1. Medical service:			
1. Syphilis			673*
2. Tabes dorsalis			95
3. Epilepsy			375
4. Hysteria			218
5. Acute apoplexies			197
6. Paralyses of ill defined origin			156†
7. Neuralgia and neuritis			122
8. Chorea			112
9. Acute encephalitis			104
10. Exophthalmic goiter			59
11. Tuberculous meningitis			29
12. Simple meningitis			27
13. Other diseases of the spinal cord			167
14. Other diseases of the nervous system			517‡
Total			2,851
2. Surgical service:			
1. Fracture of skull	243	(160 cured or improved; 83 died)	
2. Fracture of spine	19	(14 cured or improved; 5 died)	
Total	262		

* Includes primary, secondary and tertiary syphilis but not paresis or tabes.

† Includes residual hemiplegias.

‡ Infections, traumas, vascular, tumors, psychoneuroses, etc.

to ascertain the number of cases of concussion of the brain or spinal cord, it would be necessary to search the case records. It is possible, then, that in 10 per cent. of traumatic cases there should be a neurologic examination.

Of the total admissions to Bellevue Hospital, which were about 43,000 cases, in approximately 3,113 cases, or about 7 per cent, there should have been a neurologic examination.

One's first impression might be that in private practice less than 7 per cent. of patients are neurologic cases. But the Cornell Clinic in its past year admitted over 22,000 patients, and the neurologic department

examined a little over 7 per cent. of all these. So it seems reasonable to say that this proportion holds true in general practice, which would include both ambulatory and confined patients.

Consultation with the neurologic service was requested for about 875 patients. Of the total of 1,003 patients cared for or examined by the neurologic service, about 13 per cent. were admitted to the ward from outside sources. Thus, as at present organized, the neurologic service took care of about 28 per cent. of the neurologic cases in the hospital. It is not implied that the other services in the hospital were

TABLE 5.—*Chief Causes of Conditions Found in Patients*

	Directly or Immediately Affecting the Central Nervous System	Resulting in Nervous System Complications
1. Hereditary:		
Wilson's disease	1	
Friedreich's ataxia	1	
2. Tumor	54	7
3. Vascular accident	62	4
4. Environmental and [illegible]	71	
5. Congenital mental or physical defects	87	
6. Toxic (diet, metabolism, poisons)	107	
7. Unknown, including multiple sclerosis, amyotrophic lateral sclerosis, muscular dystrophy and atrophy, epilepsy	144	
8. Mechanical and physical trauma	138	26
9. Infection	76	305
	743	342
	342	
Total	1,085*	

* The total of 1,085 is somewhat more than the number of patients seen. This difference is due to the fact that in some patients two or more causal factors were of equal importance.

dependent on the neurologic service for either diagnosis or treatment of many of the neurologic cases. But from an ideal standpoint, the neurologic service should be able to take care of every case falling within its scope. Furthermore, other services in the hospital should realize, both for the sake of science and for the sake of the patient, that a neurologic survey should be given whenever indicated.

To perform this ideal function, the Bellevue Hospital neurologic service should have wards with three times the present capacity.

In Table 5, several points are of interest. In the first place, it is rare to find a patient with so-called

hereditary disease in which there seems to be any hereditary factor.

In the only instances in which so-called hereditary disease occurred in other members of the family, the disease was present in siblings. It is not intended to assert that there is no such thing as hereditary disease, but it seems likely that "hereditary" nervous diseases are no more hereditary than "hereditary" cancer or tuberculosis. It would be more accurate to classify certain "hereditary" diseases as of embryonic origin and due to some specific defect in the germ plasm, of unknown cause.

The next thing of importance is the fact that in 1,003 neurologic cases, there were only eighty-seven cases in which congenital mental or physical defect could be regarded as an important factor in the conditions presented.

In dispensary practice, there would be found a larger proportion of congenital mental or physical defects. It is my impression that the commonest sort of congenital physical defect lies in an instability of the vegetative nervous system and the endocrines.

Conditions in which the causes are unknown totaled 144 cases, or about 14 per cent. of all the patients. This group of conditions should be subject to investigation.

Eliminating the cases in which congenital defects were the chief causes, and cases in which the etiology is unknown—a total of 23 per cent. of all patients—we come to the cases in which causal conditions are to some extent under medical control. The proportionate frequency of the various causes in this group suggests what things should be emphasized in teaching medical students. Clinical rarities and obscure problems in neurophysiology and neuro-anatomy seize the attention of the average instructor. But when one finds that 7 per cent. of all medical cases are neurologic, and that three fourths of the neurologic cases are linked with common medical problems, it seems wise to insist that in teaching neurology to undergraduates the instructors curb their enthusiasm for problems that should be of interest to neurologists only.

As might be expected, the brain is most subject to insult. These insults range from psychic, through infections, toxins, vascular accidents and tumors, down to physical trauma. The vegetative nervous system

and endocrines have received a great deal of attention in the last decade. Their derangements are relatively of minor importance in patients seen by the Bellevue neurologic service, in that only seventy-three cases, or 7 per cent., presented diseases of these systems. However, the reverse is true in dispensary practice. I have no accurate figures available, but it is my impression that at least one third of dispensary neurologic patients offer problems in relation to the functions of the endocrines and the vegetative nervous system. With reference to the teaching of neural anatomy, since the statistics show (1) that over one half of all organic cases seen were represented by only seventeen different conditions, and (2) that three fourths of the neurologic

TABLE 6.—*Site of Lesion Producing the Condition*

1. Peripheral nervous system	106
2. Cord	186
3. Brain	273
4. Vegetative nervous system (includes vasomotor)	15
5. Endocrines and vegetative nervous system	18
6. Mixed:	
a. Cord and brain	88
b. Cord and nerves	32
c. Cord and vegetative nervous system	2
d. Brain and vegetative nervous system	18
7. Not of central nervous system	55
8. Epilepsy	60
9. Psychic	88
10. Psychic and vegetative nervous system	20
11. Psychic and organic	30
12. Ill defined or unknown	12
Total	1,003

cases have relation to major problems in internal medicine, it seems safe to assert that it is a mistake to confuse the undergraduate with a mass of fine details. He does not need to know anything about the bundle of Vicq d'Azyr, how many nuclei there are in the thalamus or the details of the *minor* association tracts linking the cortex, corpora striata, pons, cerebellum and cord.

There is one matter of importance that is not obvious in the statistics tabulated in this paper. If the neurologic service dealt with a larger proportion of psychoneurotic or psychiatric cases, it would be manifest that proper estimation of the patient's personality is of fundamental importance in properly evaluating his symptoms. Ability to do this depends on more than being told to remember the point. Every neuropsychia-

trist has abundant experience with patients whose complaints have been wrongly judged by practitioners in other branches of medicine. Time and again, organic cases have been regarded for months or even years as "hysterical." The reverse is equally true. Too little attention is paid to this important part of diagnosis. It should be drummed into the head of every undergraduate. Not only do patients suffer needlessly because their physicians err in estimating the psychologic equation, but the flood of quacks is to a great extent maintained by persons whose physicians have not properly handled them as human beings.

TABLE 7.—*Classification of Ward Patients*

Class	No.	Comment
A. Cases for diagnosis and hospital treatment	174	Full hospital facilities; keep up from two to three months
B. Cases for diagnosis and needing only custodial care	48	After diagnosis, refer home or to an institution; keep from one to two weeks
C. Cases for diagnosis and ambulatory treatment	61	After diagnosis, refer home; keep from one to two weeks
D. Cases incurable, confined to the home, and in which economic rehabilitation could be effected by training in handicrafts and then sale of products	28	After diagnosis, refer home or for training in a separate ward or institution
E. Acute cases *.....	61 (20%)	Full hospital facilities; keep up from two to three months

* Included in this group are a few brain tumor cases and most of the spinal cord cases. These are acute because they reach the hospital at a time when delay in reaching a decision as to treatment would be dangerous.

The organization of a neurologic service depends on the clinical material to be handled. Table 7 gives a rough classification of the patients cared for in the ward of the Bellevue Hospital neurologic service. The table itself needs no further comment except as regards Groups B and D. Group B patients are difficult to take care of. Home surroundings are usually impossible, and even if the municipality provided visiting nurses and physicians to see such patients, their lives would be shortened. The cost of upkeep of institutions for such cases would be decreased if such institutions were adjacent to a hospital equipped to care for acute medical conditions. In this manner, when custodial patients develop acute illnesses, they could

be immediately transferred to a hospital for acute cases.

In Group D, a successful solution of the economic problem depends on providing occupation, with the aid of social service organizations. At present, Bellevue Hospital provides occupational therapy for its patients. A good many of the neurologic patients stay in the ward for one to two months. Occupational therapy is of value in maintaining the morale of the patients while they are in the hospital. But there are many patients taught a useful trade who could contribute something to their support by making and selling various products after they have left the hospital. At present, such patients are dependent on charity, either from relatives or that given by aid societies. When the sick person is the sole wage earner in his family, the whole burden is thrown on organized charity. It is impossible to estimate how many such cases there are in greater New York, but 5,000 is probably a conservative figure. A comparatively simple organization would furnish facilities for training such patients and then arrange for collecting and selling the products made by them in their homes. The administration cost of such an organization would certainly be less than the present system of giving all and allowing the patient to exist in idleness. This idleness certainly is not conducive to maintaining the self-respect and morale of the invalid. The chief obstacle to the success of such a scheme might lie in the resistance of labor unions. The unions have handicapped many of the war veterans who have been rehabilitated in various vocations and find it impossible to work in unionized trades because the unions will not admit them to membership. But this obstacle ought not to be very great, because a little publicity of the right kind would certainly silence any unions who tried to complain of the bread being taken out of the mouths of their members. In fact, a good many of the invalids who would be doing such work have already been union men in various trades, and no credit would accrue to unions that asserted opposition to unfortunates in such circumstances.

Table 8 suggests the necessity for a persistent optimism in every neurologist.

In the patient with chronic organic disease, improvement may be both subjective and objective. In cases

in which treatment will produce a beneficial physical change, the patient feels the difference, and the improvement in morale is readily understood. Often one sees individuals in whom treatment can make no difference in the objective manifestations of the disease, yet such patients will tell a physician that they feel better, that their pains do not trouble them, or that they can get around more easily. The fact that these people have been sympathetically handled and thoroughly examined has been sufficient to alleviate the results of prolonged introspection and its attendant fears and despair. The patients feel better because they do not think about their troubles. Practically, such patients have to be regarded as improved, although from the material standpoint there is no change. There is a third group of patients in whom

TABLE 8.—*Results of Treatment of Ward Patients* *

	Cured or Improved Per Cent.	Unimproved Per Cent.	Died Per Cent.
All cases	48.4	43.8	7.8
Organic	46.5	44.7	8.8
Psychoneuroses	75	25	
Psychoses	20	80†	

* Two hundred and eighty-three different patients admitted during 1921, with seventeen readmissions.

† These patients were transferred to the psychopathic service after a brief period of observation.

impairment of morale has caused them to stop using their bodies to the full extent possible. Multiple sclerosis, tabes and residual encephalitis cases illustrate this group best. After they have been in the ward for a week or two, they will be encouraged to make efforts and, as a result, learn that they are not so badly off as they feared. They will leave the hospital actually walking in a much improved way. There has been no change in the pathologic anatomy, but the function left is used to its fullest extent. In effecting improvement in chronic cases, the atmosphere of the ward is fully as important as any physical mode of treatment. A physician who treats his patients only as diagnostic problems will never be successful in the removal of what might be called "emotional overflow" symptoms. The average person demands *something* in the way of treatment. In my mind, physiotherapy produces bene-

fit in organic neurologic cases, chiefly because it is a concrete demonstration, and is something new to the average patient.

HOSPITAL ORGANIZATION

Since approximately 7 per cent. of the patients admitted to Bellevue Hospital need a neurologic survey, it seems to me that every general hospital with a capacity of 500 beds should have a neurologic service. The staff of this service should include a sufficient number of attending physicians making regular rounds to examine the patients on whom consultation is requested. There should also be one intern whose full time would be devoted to the neurologic patients. He should examine every neurologic patient and be able not only to select those needing the attention of a visiting physician, but also to direct their treatment under the supervision of the visiting physicians. There should be a separate neurologic ward in every hospital of more than 1,000 beds. The intern staff should by all means be limited to those who have completed a general medical internship. It may be well to emphasize at this point that a neurologist has a function often quite distinct from that of a psychiatrist, and to my mind it is ridiculous for a man to call himself a neuropsychiatrist when his clinical training and experience are limited to psychiatry alone.

One must be careful about throwing stones, and it is just as true that a neurologist may make costly mistakes in dealing with psychopathic patients, because his experience has not been sufficient to develop judgment nice enough to tell him just when the patient reaches the point at which he must be confined to an institution.

As for the organization of the neurologic service, the classification of patients given in Table 7 suggests that there must be a sufficient number of beds to enable patients to be kept long enough to benefit by treatment. There is nothing to be gained by having a separate neurologic ward if the patients admitted to it are kept only for diagnosis. Moreover, the period of observation necessary for a reasonably accurate diagnosis varies greatly. We have found at Bellevue that two or three weeks of careful observation may frequently be necessary before a correct diagnosis can be established. Again, we confess that, at times, our diagnostic acumen has been completely baffled in certain cases.

A former resident in our service used to account for some of the puzzling cases on the ground that the patients had failed to read the textbooks.

GENERAL CONSIDERATIONS

While it is true that a great many organic nervous diseases cannot be cured and that the course of the disease is long, these patients are just the ones to furnish material for medical research. They are important, too, because many of them, at one time or another, have been treated by cult practitioners. It is impossible to keep all chronic patients out of the hands of quacks, who secure confidence solely by making extravagant promises of relief or cure. We found that a large proportion of our chronic cases had never been properly studied and diagnosed by any one; and once these patients had been carefully examined and observed, adjusted themselves well to the fact that their diseases were incurable.

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SPASMODIC DIPLOPIA

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Double vision, either transitory or constant, is a fairly common symptom in the practice of ophthalmology and neurology. During the last five years, the literature of epidemic encephalitis has so emphasized diplopia as being a symptom of diagnostic importance that perhaps one is apt to regard it, in certain types of obscure cases, as evidence of encephalitis.

Constant diplopia is not so difficult to account for in a given patient. It is my purpose in this paper to present a small series of cases in which transitory diplopia occurred and had a significance which, I believe, is worthy of discussion.

In eliciting a history of this symptom, it is important to differentiate distinct double vision from mere blurring or indistinctness of objects. It is not uncommon to have a patient say that he sees double at times, yet on questioning it is manifest that the patient refers merely to indistinctness of objects. Of course, errors of refraction and muscular insufficiency may produce such phenomena, which one might regard as milder degrees of real diplopia. However, the characteristic of genuine transitory diplopia is that objects will suddenly appear with two images, one of them distinct and the other blurred. In the diplopia discussed in this paper the symptom occurred quite independently of use of the eyes, was sudden in onset, and would continue for a few seconds, or at times a minute or two.

REPORT OF CASES [1]

Case 1.—J. K., a man, aged 37, whose family and past history were irrelevant except that in February and March, 1923, he was ill for a month with influenza and pleurisy; there being

1. Case 1 was observed in the neurologic service of Bellevue Hospital, and Cases 3, 4 and 7 in the Cornell Clinic.

nothing at this time to suggest encephalitis, he had been perfectly well until the present illness appeared.

About the middle of August, 1923, in a period of a few days, there were (1) belching, bloating and then constipation; (2) sleeplessness; (3) restlessness and a tendency to change positions often. It was this restlessness which the patient thought was responsible for his sleeplessness. From the onset until the middle of September, these symptoms continued and were accompanied also by irritability and quite frequent transitory double vision when looking at distant objects. On examination, the only abnormalities were myotactic irritability, grossly exaggerated deep and superficial reflexes, hyperhidrosis and a slowing of the pulse rate from 88 to 66 beats a minute by ocular pressure. The serum, blood and urine were normal. Examination of the eyes showed normal pupils and fundi, no nystagmus, no refractive error and no evidence of muscle weakness.

In further investigation of the history, it was discovered that the patient had for about a year been drinking 2 or 3 quarts of strong coffee a day, that he might carry on with his long hours of work.

The sole treatment was to discontinue the use of coffee, and the patient was fully recovered by the first of October.

Case 2.—Mrs. T., aged 36, whose past history was negative except that she had always been "nervous," had what she thought was ptomain poisoning about June 25, 1923, after eating a frankfurter. No one else in the family was upset, however. There was a slight fever and then, after about two days, diarrhea without abdominal pain. Sleeplessness appeared with the diarrhea, and both symptoms persisted to the time of her first visit, July 6. From July 2 there was considerable headache. For nearly a year there had been numbness in the finger tips, worse after sewing and troublesome when she went to bed. For several weeks she had seen double at times. This double vision was in no way related to the use of her eyes. It came suddenly, lasted a minute or two, and was evident when looking at distant objects. She had no nose or throat trouble. Her oculist stated that no glasses were needed.

The patient was stout. The systolic blood pressure was 130; diastolic, 70. There was no evidence of thoracic or abdominal disease. The nervous system showed no abnormality other than exaggerated tendon reflexes and a slowing of the pulse from 84 to 60 beats a minute by ocular pressure. The urine, temperature and fundi were normal.

Treatment consisted of thyroid extract, ¼ grain (0.016 gm.), and phenobarbital (luminal) ¾ grain (0.05 gm.), twice a day; also 4 ounces (120 c.c.) of sherry wine in warm water on retiring and sleeping on a high pillow were prescribed.

On July 13, the patient stated that her sleep had been normal from the first night of treatment. The diarrhea

stopped after July 7, and the nervousness and numbness in the fingers had ceased. The pulse was 78; diplopia had occurred twice since July 6, and lasted only a minute or so.

A letter from the patient, October 26, stated that there was no diplopia during the summer, but that the symptom had occurred twice between October 15 and October 26. The paresthesias in the finger tips occurred occasionally. The patient did not report, and the cause of the recurrence of diplopia can only be guessed at.

Case 3.—Mrs. P., aged 45, seen June 29, 1923, whose past history was irrelevant and in whom menstruation was unchanged, for a year had complained of dizziness and a feeling of weight at the back of the head, especially when lying on the left side. During this year there had developed constipation, belching and a frequency of urination, which was influenced by what she called "nervous tension." For six months she had noticed brief attacks of double vision, occurring perhaps twice a week. This symptom appeared suddenly, and was not related to the use of the eyes.

Physical examination showed normal blood pressure, a coated tongue and no evidence of disease of the thoracic or abdominal viscera. Special examination of the ear, nose and throat, including transillumination of the sinuses, was negative. Ophthalmologic examination showed a slight astigmatism with normal acuity and muscular balance. The patient's glasses were stated to be proper for her refractive error. Examination of the nervous system showed nothing abnormal, and ocular pressure caused no change in the pulse rate.

The patient was given a prescription containing aloin $\frac{1}{10}$ grain (0.0065 gm.); atropin sulphate $\frac{1}{150}$ grain, and phenobarbital sodium, $\frac{1}{4}$ grain (0.016 gm.), to be taken three times a day.

July 13, she stated that her dizziness and the gastrointestinal symptoms were better. The diplopia attacks were shorter in duration.

July 27, the patient stated that her symptoms had all disappeared.

September 7, the patient reported again and asserted that there had been no recurrence of the symptoms. The menses were still normal.

Case 4.—J. M., a man, aged 34, seen in August, 1922, had been sickly and had frequent attacks of croup, when a child. For the last seven years he had complained of deafness and tinnitus in the right ear. There was also a myopia, with glasses giving correction to normal vision. For about six months he had been having sudden "spells," coming at irregular intervals. Each spell was ushered in with a sudden, intense vertigo and a terrific noise in the right ear. Pallor, vomiting and a cold perspiration came next. The acute symptoms diminished in a few moments, but each spell was

followed by a brief attack of diarrhea. Since these spells appeared, he had had constipation. In none of the attacks was there any double vision, but on several occasions he had noticed, while at his work as a butcher, that some objects would suddenly seem distinctly double. He did not know of any exciting cause for these spells, but had observed that for a day or more before each attack his appetite was poor and that for a few hours previous he was slightly dizzy.

The patient was of a status lymphaticus type. There was no disease of the thoracic or abdominal viscera. There was marked dermographia and a respiratory arrhythmia of the pulse, which, however, was not affected by ocular pressure. Otologic examination showed a partial degeneration of the right auditory nerve. The patient was given atropin sulphate, $\frac{1}{150}$ grain, and phenobarbital, $\frac{1}{4}$ grain (0.05gm.), to be taken twice a day.

He reported in October, 1922, that his attacks and constipation had ceased. Medication was discontinued.

In August, 1923, he stated that none of his symptoms had reappeared. The tinnitus and deafness in the right ear, however, remained unchanged.

In January, 1924, the patient stated that for about two weeks he had been having slight, brief attacks of dizziness, which were accompanied by an increase of the tinnitus in the right ear and moderate sweating. For three months he had been employed delivering milk, his hours of work being from 3 to 10 a. m. He had again become careless about his bowel movements and finally, about two weeks before the reappearance of the dizzy spells, constipation developed. The same treatment given in 1922 afforded relief again.

Case 5.—Dr. E. L., a man, aged 32, whose past history was negative, for about a year had been under extra strain in his work as a surgical intern. In April, 1923, a slight left frontal sinusitis suddenly appeared. It was neither preceded nor followed by any symptoms to suggest infection of the nasopharynx, and cleared up in a few days. About a week later, epigastric pain, belching and constipation developed. Next, frequent sudden attacks of dizziness, fatigability and an esophagospasm developed. One day in June, while operating, the patient suddenly became very dizzy, so that he had to steady himself. He was able to continue with the operation, which was for a radical removal of the breast, until, owing to sudden diplopia, he clamped a nerve in the brachial plexus instead of an adjacent artery. During the next few weeks all symptoms increased.

In July, 1923, atropin sulphate, $\frac{1}{100}$ grain (0.00065 gm.), twice a day, stopped all the symptoms.

During August and September, while he was taking a vacation, the gastro-intestinal symptoms recurred. There was bloating after meals, and there were occasional attacks

of pain and tenderness over the region of the appendix. Diets were only partly effective, and atropin did not seem to help. On one occasion there was an attack of sudden dizziness, with a slowing of the pulse to 50 beats a minute. This state lasted about an hour.

Gastro-intestinal roentgen-ray examination showed no organic lesion.

Case 6.—G. B., a man, aged 38, whose past history was negative except for overwork for the last four or five years, began to have constant tinnitus in the right ear in July, 1921. About 5 o'clock one morning in August, he awoke in a profuse sweat. He felt nauseated, and there was an intense desire to defecate. He hurried to the bathroom and was suddenly seized with vertigo so violent that he fell to the floor. He vomited and lost control of both sphincters. After about ten minutes he was able to get back to bed and stayed there for the rest of the day, feeling prostrated.

From this time on there was marked constitpation, with hard, dark-colored, scanty movements. There was a gradual loss of weight.

Between June and September, 1922, the patient had perhaps twenty sudden attacks of dizziness, lasting five or ten minutes. They usually occurred toward the end of the day. The onset was sudden and followed by belching, sweating and weakness for about an hour, and occasionally a desire to defecate. Some attacks were ushered in with gastric distress and belching, with dizziness following in a few seconds. On one occasion, while the patient was walking in the street, there was distinct double vision in the horizontal plane for both near and distant objects. This lasted about half a minute, and was not accompanied by any other symptom.

The patient and his wife had both noticed that a dizzy attack was usually preceded by an increase in constipation and a heaviness of breath in the morning.

Early in October, two severe attacks occurred, three days apart. In the second attack the patient was thrown to the sidewalk and struck his head forcibly. He was dazed for a moment, but was able, with help, to walk to his home, a block away. Examination made an hour after this attack showed nystagmus to the right, an increase of deep reflexes on the right side, a questionable right plantar response, and a positive Romberg sign. In addition, the patient was hypotrichotic, had a faulty posture, with a ptosed abdomen, and there was a blowing systolic heart murmur. The Wassermann reaction was negative; the blood pressure, blood count and urine were normal.

The patient was ordered to take a vacation. Diet and exercise were prescribed. In addition, he was given aloin, 1/10 grain (0.0065 gm.) to be taken three times a day. He was away for a month, during which time he had four slight attacks of dizziness. In one, objects moved rapidly to the

right, in another the only symptoms were dizziness and double vision for a few seconds. Double vision occurred once independently of any other phenomena.

By December, the patient had gained 5 pounds (2.3 kg.), the bowels were regular, and except for three mild attacks of belching and sweating, he had no complaints.

Otologic examination in October showed an irritability of the vestibular apparatus and a slight nerve deafness on the right side. The nystagmus and pyramidal tract phenomena that were observed on the first examination continued for several days. The nystagmus disappeared last, and toward the end could be elicited only when the patient was standing. The heart murmur had disappeared.

The patient was well until the summer of 1923, when on two occasions constipation developed, and there were three or four slight attacks of dizziness. Lactose and *Bacillus acidophilus* culture were effective in relieving the constipation.

Early in October, 1923, the constipation appeared again, and for several days the patient did nothing about it. One afternoon he went to a football game without having taken any lunch. At 5 o'clock he ate a frankfurter. At 6:15, when nearly home, he suddenly became very dizzy and staggered as though intoxicated. When he got into the house he had to lie down. He belched continuously, his abdomen felt bloated, and he perspired profusely. Examination showed occasional extrasystoles and a marked respiratory irregularity of the pulse. Ocular pressure reduced the pulse from 90 to 66 beats a minute. The skin was cold and moist, and there was a distinct dermographia. Palpation of the abdomen revealed a mass in the upper right quadrant. It was not tender, it moved with deep inspiration, and it seemed to run up under the border of the liver.

Gastro-intestinal fluoroscopic examination showed hypertonicity of the viscera and a kink at the hepatic flexure, causing a partial obstruction and slowing the passage of intestinal contents.

Exercise to correct the faulty posture, a roughage diet, and abdominal massage were prescribed.

At the present time (January, 1924), the patient is getting along well. He has been instructed how to watch for and detect a reappearance of the mass in the upper right quadrant, which is due to an accumulation of the contents of the colon at the hepatic flexure.

Case 7.—Miss R. B., aged 42, seen in February, 1923, whose past history was negative, during the preceding half year had suffered from constipation and had lost about 10 pounds (4.5 kg.). She recently began to have pain in the left shoulder, which increased and radiated to the back of the neck. It made it difficult for her to raise her arm.

Physical and neurologic examination showed nothing in particular except tenderness over the cervical region of the

spine, left posterior triangle of the neck, in the left shoulder joint, and in the left deltoid and triceps muscles. There was slight wasting of the deltoid. Blood count, blood analysis and urinalysis were negative. A roentgenogram of the cervical and upper dorsal spine showed slight ostearthritic changes. Gastro-intestinal roentgen-ray examination showed evidence of diverticulitis in the descending colon and sigmoid.

Aloin and atropin relieved the gastro-intestinal symptoms and reduced the pain in the shoulder for two or three months. Thyroid extract, ¼ grain (0.016 gm.), twice a day was then prescribed because dryness of the skin, falling of the hair from the eyebrows and brittleness of the finger nails appeared. Further improvement of the shoulder pain followed. Finally, the patient was advised to have vaccine treatment, and after a three months' course of a stock vaccine of *Streptococcus viridans* and *Streptococcus fecalis,* the shoulder pain was entirely relieved. However, the gastro-intestinal symptoms recurred from time to time, although colon irrigation and *Bacillus acidophilus* culture were helpful in reducing the frequency and severity of exacerbations of the digestive disturbances. In November, 1923, the patient observed that two or three times she had noticed that objects appeared double for a moment or two.

The first time the double vision occurred was within a half hour after taking a bismuth meal. The double vision itself was described as being a blurring of near objects, but objects 5 feet or more away seemed double in the horizontal plane.

In this case the significance of the double vision is open to question, because the patient is a manuscript reader and for several years has had to wear glasses. Her eyes were examined and the glasses which she had been wearing were found to be proper for her refractive error. The patient volunteered, however, that she did not think her work had anything to do with the double vision, because at no time did it occur when she was at work. Furthermore, she thought that something about her digestion was responsible, because on the days when she had the double vision she had been troubled with belching.

REVIEW OF CLINICAL MATERIAL

Three of the patients were women; four were men. It is interesting to note what conditions might be regarded as predisposing. Of the women, two were approaching the menopause, although in neither had there been any changes in menstruation; the third had always been "nervous." Of the four men, three were definitely hypotrichotic, and one of them gave a history of frequent attacks of croup as a child; in the fourth man, Patient 1, there was no constitutional state that might be regarded as predisposing.

Digestive disturbances of one sort or another were present in every instance. In Cases 1, 2 and 6, the presenting symptoms seemed to be related to some article of food ingested. In Cases 3, 4 and 7, there was nothing definite to suggest any particular article of food as a cause for the various occasions in which symptoms appeared. In Case 5, fatigue or "nervous exhaustion" seemed to be a precipitating factor. In Cases 4 and 6 there was chronic ear disease.

In surveying the group of cases, certain symptoms and signs pointed to the fact that the autonomic part of the vegetative nervous system was overactive. Spastic constipation, gastric unrest and eructations, respiratory arrhythmia strongly positive oculocardiac reflex responses, hyperhidrosis, vasovagal attacks, esophagospasm and frequency of urination may be mentioned. The predisposing factors in the women were of a sort that are often associated with an instability or hyperexcitability of the vegetative nervous system. Endocrine imbalance and metabolic disturbances, which are common in such instances, might be regarded as part and parcel of an upset vegetative mechanism. In the men, the hypotrichosis is a sort of "outward and visible sign" frequently associated with inadequately balanced vegetative and endocrine systems. The symptoms observed, which were referable to an overactive autonomic, would, if combined in one person, form a composite clinical picture that would be called vagotonia. Of course, one may find in any sick person one or more symptoms that suggest autonomic overactivity, and an enthusiastic imagination might lead one to wrongly call such a case vagotonic. However, in the patients described here, not only was there an absence of prominent symptoms of other nature, but in each patient there was at one time or another a sufficient variety of vagotonic phenomena to warrant the view that these patients really had vagotonic syndromes.

The treatment of the various cases is difficult of analysis because of the diversity of measures used. In one instance the cause of the symptoms seemed to be excessive consumption of coffee, and discontinuing the beverage was sufficient to bring about a cure. Theoretically, atropin alone should have been used in every case, if one were to conduct pharmacologic experiments. But, unfortunately, discretion demands that a patient be relieved as quickly as possible, and in most of the

cases here reported, more than one measure was indicated.

Caffein is known to be a vagus stimulant, and in the "caffein poisoned" man, withdrawal of coffee was mathematically the same as using atropin to diminish vagus tone. In Case 5, atropin alone did have a temporary effect. In Case 4, atropin and phenobarbital were used; I think it unlikely that phenobarbital was solely responsible for the relief of symptoms, and that therefore atropin should be given some credit.

COMMENT

There are many causes for transitory diplopia. It may occur in multiple sclerosis, during the attacks of Ménière's disease, and in lesions affecting the function of the ocular muscles—basal meningitis, intrapontile and extrapontile tumors, vascular lesions of the pons and midbrain, epidemic encephalitis, and pressure on one or more of the nerves supplying the eye muscles. Infection of the teeth and cranial sinuses may produce paralysis of the ocular muscles, and some authors state that reflex spasm of the ocular muscles to produce transient binocular diplopia may be due to similar infections. In the latter event, the transitory diplopia is usually more distinct than the double vision in partial paralytic involvement of the ocular muscles. The diplopia in the series of cases here presented cannot be explained by any of the foregoing causes. Eppinger and Hess state that vagotonic persons frequently have anomalies of muscular control of the eyes. They do not describe or illustrate what they mean, but the statement suggests that in some way the cerebral portion of the autonomic may at times, when the entire autonomic is in a state of overactivity, influence momentarily the tonus of the muscles supplied by the oculomotor nerves. Perhaps this sort of phenomenon might be regarded as analogous to the extrasystoles occurring in "vagotonic" persons. In the so-called reflex accommodation spasm, when sinus or dental infection is an associated condition, the mechanism is evidently in the nature of a local reflex.

Although in this series of cases it might be maintained that the diplopia is a vagotonic phenomenon, since it is accompanied by other clinical features characteristic of vagotonia, there are certain questions of physiology that need answering before such an hypo-

thesis can be accepted. In particular, attention must be paid to the two cases in which there was ear disease, with symptoms quite similar to those described in Ménière's syndrome.

In organic ear disease, tinnitus and vertigo are not difficult to understand. But there are instances of Ménière's syndrome in which not only temporary vasomotor disturbance in the labyrinth but often "angioneurotic" increase in the perilymph have been regarded as causal. It has been stated [2] that "in certain debilitated states of the nervous system, symptoms closely resembling, if not identical with, Ménière's disease may occur." Seasickness illustrates the relation between the vestibular mechanism and that portion of the vegetative nervous system stimulating gastro-intestinal musculature. Gowers,[3] discusses the frequent difficulty in distinguishing vasovagal attacks from Ménière's syndrome. Biehl [4] cites several cases of tinnitus in which the symptom was relieved by drugs depressing the peripheral portion of the autonomic, and concludes that tinnitus may at times be regarded as evidence of vagotonia and is of vasomotor origin. The value of epinephrin in treating ear disease is reported by several otologists. Duke [5] reports two cases of Ménière's syndrome seemingly caused by allergy and cured by the administration of epinephrin. (Did not the epinephrin act really to reduce the vagotonic state by increasing sympathetic tone?) It seems reasonable, therefore, to maintain that conditions affecting the vagus mechanism may cause labyrinthine or vestibular symptoms.

In the two cases of ear disease in my series, it would indeed be rash to assume that the deafness originated in a changed vasomotor state due to autonomic influence; but it is not unreasonable to consider that the presenting symptoms were to some degree conditioned by the state of the auditory apparatus.

Diplopia is known to occur in Ménière's disease in association with the attacks of vertigo, and in these instances may be accounted for by a spread of impulses from the auditory pathways to their connections with the posterior longitudinal bundle, which controls ocular

2. Osler: Practice of Medicine **7**: 568, 1910.
3. Gowers: Borderline of Epilepsy, 1907.
4. Biehl: Vagotonia in Relation to the Ear, München. med. Wchnschr. **67**: 1263 (Oct. 29) 1920.
5. Duke, W. W.: Ménière's Syndrome Caused by Allergy, J. A. M. A. **81**: 2179 (Dec. 29) 1923.

muscle coordination. However, the diplopia in my cases will not admit this explanation. In the first place, only two of my patients had symptoms suggesting Ménière's syndrome, and only once, in them, was diplopia accompanied by vertigo. On all other occasions, with these patients, the diplopia occurred alone. In Case 5, diplopia occurred once and was then associated with vertigo; but that patient had nothing to suggest ear disease. In the other patients, the diplopia was never associated with any symptom suggesting vestibular or labyrinthine disturbances.

The diplopia in my series of cases seems to call for another explanation. On the one hand, the relationship between the vagus and the auditory nerve functions, and, on the other hand, the occasional occurrence of diplopia in Ménière's syndrome are somewhat suggestive of the hypothesis that an undue state of vagus irritation might itself at times provoke diplopia.

Timme [6] states that in certain types of pituitary disturbance there may occur a temporary swelling of the pituitary body so that there is pressure on the walls of the cavernous sinus, and that during the headaches there may be present ophthalmoplegic phenomena which will include diplopia, strabismus, ptosis or pupillary abnormalities. The clinical evidence in my series of cases obviously excludes this explanation for the diplopia.

Finally, "muscular insufficiency" or refractive errors cannot account for the diplopia. Ordinarily, accommodation spasm associated with refractive errors produces diplopia on distant vision; but such diplopia is not so fleeting as that described in this paper. Of the seven patients, three had normal vision; two wore glasses that properly corrected their refractive errors; one with astigmatism had her glasses refitted without relief, and one was myopic, the refractive error being corrected with glasses. Of course, eye strain has been held responsible for some cases of gastro-intestinal disturbance; but such an explanation for the vagotonic phenomena described in this series of cases would, in my opinion, indicate a lack of clinical perspective. If gastro-intestinal symptoms can be reflexly due to eye strain, one might on the analogy of "what is food for the goose is sauce for the gander" think that gastro-

6. Timme: "Evolution Status Thymico Lymphaticus," New York M. J. **114**: 12 (July 6) 1921.

intestinal disturbances might reflexly produce some disturbances of ocular function. Perhaps the statement of Eppinger and Hess, that vagotonia may present anomalies of muscular control of the eyes, supports such an hypothesis. Accommodation spasm has been mentioned as one of the manifestations of vagotonia, but in my experience this accommodation spasm is more persistent, and I have not seen it producing the sort of diplopia described in this paper.

It is my belief that the diplopia itself in this series of cases should be considered as a manifestation of an irritated autonomic mechanism, and that it is analogous to the occurrence of extrasystoles in "vagotonic" persons. I have no statistics indicating how often the symptom occurs in "vagotonic" states, but it is my impression that it is present in perhaps 10 per cent. of such cases. Perhaps the incidence is greater, but this can be determined only by further investigation of case histories. It is, I think, possible that the symptom might occur in the so-called vegetative neuroses when the clinical picture is not characteristic of vagotonia. It must be kept in mind that recent clinical observations have led us away from the formal concepts of Eppinger and Hess, and that individuals should be considered not as vagotonic or sympatheticotonic, but rather as having hypertonia or an abnormal irritability of the entire vegetative nervous system.

From the clinical point of view, the diplopia described in this series of cases has a diagnostic significance. Because of this symptom, one patient was considered by his physician to be suffering from acute epidemic encephalitis. Patient 3 had been refitted with glasses without any benefit, and Patient 7 was about to consult an ophthalmologist. Another lesson from these cases is the importance of a complete and accurate history.

Perhaps this type of diplopia should have a separate designation. Transitory diplopia is not quite descriptive. Spasmodic diplopia might be a better term. It implies its transitory nature, yet differentiates it from the transitory diplopia due to weakness of the ocular muscles caused by any one of a variety of conditions. Finally, the term indicates what I believe is the nature of the functional disturbance—a temporary overaction of certain muscles supplied by the oculomotor nerves. Bearing on this point were the facts that the diplopia was always described by the patients as being in the

horizonal plane and most noticeable on relaxed or distant vision or when there was no effort at close accommodation. It is not easy to establish whether the spasm is one of accommodation involving the ciliary muscle, or of convergence involving the internal rectus muscle, or of both. The usual accommodation spasm described as occurring with vagotonic states might be regarded as analogous to the paroxysmal tachycardia that has been attributed to vagus activity, whereas the diplopia described in this paper is rather analogous to the cardiac extrasystoles. The fleeting diplopia in these cases never occurred at a time when the patient could be tested for muscular insufficiency. It would be helpful to have such patients carry about with them a bit of colored glass so that they could observe the position of the false image when the diplopia was present.

SUMMARY

1. Seven patients had symptoms referable to an overactive vagus function, and all had a transitory diplopia not easily explained by the usual causes and not related to use of the eyes.

2. The diplopia is best explained as a manifestation of the state called vagotonia, is analogous to the cardiac extrasystoles observed in vagotonic states, and is due to a brief spasm of certain muscles supplied by the oculomotor nerves.

3. The term spasmodic diplopia is offered as best descriptive of the type of diplopia observed in the series of cases reported.

116 East Sixty-Third Street.

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ACUTE MULTIPLE SCLEROSIS: AN UNUSUAL CASE.

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Dr. Foster Kennedy has remarked upon the fact that acute multiple sclerosis is becoming more common than formerly was the case. In 1921 the Neurological Service of Bellevue Hospital saw twenty-one cases of multiple sclerosis. Of these, five were of the acute type.

The following case presents a rather unusual picture.

Case I.—J. M., male, aged twenty-one; admitted September 28, 1921. The parents were born in northern Italy and are still living and in good health. One brother and one sister are in good health. Ten children died in early childhood and questioning revealed no evidence of there having been syphilitic symptoms in them. The mother had no miscarriages. Examination of the father and mother revealed no clinical evidences of syphilis. The patient is a laborer and had always been in good health. He denied venereal disease. His habits were temperate. Tobacco was used occasionally and until 1919 he drank beer and since then about two glasses of homemade wine a day.

In February, 1919, the patient fell about ten feet, striking his back and occiput. He was unconscious for a few minutes. Ensuing headache and backache kept him at home for two days. He then returned to work but felt so weak that after a few days he quit and entered the Neurological Institute. He

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stayed there two weeks without relief. Then he entered St. Vincent's Hospital, complaining of headache, vertigo, vomiting, staggering gait, and general weakness. After two weeks in this hospital diplopia appeared. In two months all his symptoms but the staggering gait cleared up. For a year he felt well. The gait difficulty gradually disappeared and the only symptoms were occasional paresthesia in the hands and feet, and a bit of urgency in urination. These symptoms occurred about once a month and lasted two or three days. In the summer of 1920 headache, vertigo, vomiting and staggering gait recurred. The attack this time cleared up in two or three weeks. In September, 1921, a dull, constant, occipital headache appeared. This was intensified by motion. There were additional disorders of objective vertigo, vomiting without nausea (usually after eating), numbness in the hands and ataxic gait. Within a few days the symptoms increased to such an extent that the patient had to enter the hospital. There were no bladder symptoms.

The patient was a well developed young man, weighing about 160 pounds and was five feet eight inches tall. His skin was clear, hair light brown, body hair normal in distribution. He seemed to be suffering acutely and was unable to turn about in bed because any movement produced vomiting, unaccompanied by nausea. The general physical examination revealed no abnormalities.

The pupils were equal and reacted normally. The right disc was blurred, the left pale in the temporal half. There was nystagmus in all planes and slight peripheral weakness of the left side of the face. The other cranial nerves, except the eighth, showed no abnormalities. Vestibular tests were impossible to judge because the patient could not stand having them performed properly.

The speech was of a cerebellar type. The gait

was broad based and ataxic, with deviation to the right and a tendency to retropulsion. The Romberg sign was marked and brought out the retropulsion. Finger to nose and heel to knee tests demonstrated exaggerated intention tremor. There was adiadokokinesis on both the right and left of equal degree, and underlying the intention tremor could be discerned a tendency to past point.

On the left side of the entire body there was a moderate degree of diminution of pain and temperature sensibility. Touch and position sense were normal.

The tendon reflexes were definitely increased on the right and the abdominals were absent. Weak ankle clonus, Babinski, Oppenheim and Hoffman were found on the right. The left plantar reflex showed a tendency toward extension.

The pulse was from 90 to 100. Serology of the blood and spinal fluid was entirely negative. The blood count was normal.

The patient was in the hospital for five weeks. Treatment of silver salvarsan twice weekly, in doses of 0.2 gram, was given.

While under observation there were frequent, rapid and marked fluctuations in the clinical picture, and several new symptoms appeared. Salivation with a thick, tenacious sputum, nodding tremor of the head, diplopia on looking to the right, both urgency and difficulty in starting urination, and paresthesia of the left face and forehead were noted. On November 2nd, the patient was discharged, free of symptoms and showing none of the signs found at the time of his admission except slight increase in tendon reflexes on the right, diminished abdominal reflexes and sluggish plantar responses. The patient reported back to the ward for examination in December, 1921, and January, 1922. Except for occasional paresthesia in the hands there were no symptoms or signs.

SUMMARY OF THE CASE.

A young man, previously in good health, has within thirty months three episodes suggesting acute cerebellar and pontine lesions. In the intervals he is free of symptoms, except for paresthesia in the hands and a little bladder difficulty, and he works as a laborer.

The third episode lasts nearly two months. During the attack, nystagmus, diplopia, vertigo, impaired bladder control, speech difficulty, ataxic gait, bilateral pyramidal tract signs (more on the right) and a left sided diminution of pain and temperature sensibility were noted. These symptoms were the most prominent and persisted longest.

DISCUSSION.

The diagnosis in this case lies between tumor of the cerebellum or pons, thrombosis of the posterior inferior cerebellar artery (atypical, to be sure), angioneurotic edema of the pons and cerebellum, and multiple sclerosis.

The clinical course and multiplicity of symptoms rule out tumor of any region. Thrombosis of a luetic origin finds no supporting basis in this case. In any event, the gradual increase in severity and the variety of symptoms, in each of the three episodes, precludes thrombosis. Angioneurotic edema is excluded as a possibility for the same reasons. Solitary gumma, which would be a rarity even if syphilis was present, can be ruled out.

Multiple sclerosis, then, is the logical diagnosis. There may be difference of opinion as to whether this case should be called an acute one. However, in the volcanic onrush of symptoms and equally rapid recovery, we have a picture not at all conforming to the accepted ideas of multiple sclerosis. It is true that the literature cites occasional acute cases. But this case differs considerably from the textbook acute case. Possibly the experience of the

Neurological Service of Bellevue Hospital has been too rich in unusual forms of multiple sclerosis. But in any event, attention should be called to the comparative frequency of these acute types of the disease.

I am indebted to Dr. Foster Kennedy for permission to publish this report.

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"ANTIGRAVITY MUSCLES"

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The application of the phrase "antigravity muscles" to certain extensor muscles which maintain certain animals in a standing position appears in an article by Sherrington in 1910 (1). His statement applied to decerebrated cats and dogs. He stated (p. 2): "The extensor muscles of all four limbs, the dorsal muscles of the back, neck, and tail, the retractors of the head, and the elevators of the lower jaw, are all in harmoniously coördinated steady tonic contraction." He continues (p. 3): "In the limbs they keep the limb from yielding under its own weight and under that of the body superimposed. In neck and head they keep those parts from falling; similarly in the tail they keep it from dropping, and in the head they keep the lower jaw in apposition to the upper, so that the mouth is closed.

"The muscles exhibiting the steady contraction executing this general reflex, although they are widely distributed in the body, compose nevertheless a homogeneous system. They all by their contraction contribute to one common result. They each and all, by contracting, *counteract gravity*[1] in the *ordinary erect position* usual to the animal, the *ususstatus*[2]."

The term was therefore specifically applied to the ususstatus or usual standing position of the cat and dog. With the use of the phrase in this manner I am entirely in accord. However, a number of authors (2) (myself included) (4) have since that time used

[1] Italics by W. M. K.
[2] Italics by C. S. S.

the term less precisely, without indicating the animal and that it was in its usustatus, so that it has come to mean a definite group of muscles, much as one would speak of the flexors or extensors, rather than a group of muscles active in certain animals under certain circumstances. A certain group of muscles is understood irrespective of the animal and its posture. Further, the word " usustatus " has not been added so that the phrase has come to suggest that these muscles counteract gravity *under all circumstances,* and that they are the only muscles which counteract gravity, which is not true. Furthermore, these muscles obviously have other functions which indicates that the label " antigravity " is too exclusive. Thus the original idea of Sherrington has been distorted.

In order to illustrate this, I shall consider three questions:

1. Whether in all animals these muscles are the only ones which counteract gravity and are hence deserving of the term " antigravity muscles " to the exclusion of other muscles.

2. Whether the corresponding opposite and antagonistic muscles are not equally " antigravity muscles " under certain conditions.

3. Whether these so-called " antigravity muscles " have not other functions equally important which are omitted by the application of the label " antigravity."

In reply to these first two questions, I would simply call attention to the sloth which hangs by its claws from the branches of trees, to certain lizards, to anteaters, squirrels, and opossums. These animals obviously counteract gravity by their limb and axis flexors at times, *i.e.,* are upside down. Furthermore, a circus acrobat hanging by his feet will use limb flexors, not extensors, to counteract gravity.

A term so important in its relation to posture and motility should obviously be applicable to all animals. Unless the term " usustatus " be used and the animal specified, the term will be often grossly incorrect. The antigravity muscles of the sloth are not those of the cat or dog, nor are those of an acrobat suspended by his feet those of his " usustatus."

The third question may be answered in the affirmative by simply realizing that these muscles are concerned in progression forward or backward as well as in counteracting gravity.

Now progression preceded the counteracting of gravity, for fish progress but do not have to counteract gravity. The dorsal as well as the ventral trunk musculature of fish have a quite definite function of producing progression. The counteracting of gravity appeared when terrestial life appeared. Hence the antigravity function of the extensors of the trunk was secondary to its progression function. In the course of development, as Vallois(5) has pointed out, the

dorsal trunk muscles came to have the function of support and became attached to the vertebrae (urodeles), as is not the case in animals who use the muscles only for progression (fish). In the lower types of animals, as, for example, fish, the vertebral column acts as a spring which counteracts the curving action of the muscles; in other words, the vertebral column is opposed to the action of the trunk muscles. On the other hand, " pari passu " with the assumption by the limbs of the function of progression in tetrapodal land animals and the vertebral column of the function of support, the dorsal muscles become more and more firmly attached to the vertebrae (5) (p. 510). The vertebral column and dorsal axial muscles come to act dorsoventrally rather than laterally. However, in all animals, man included, these muscles retain some influence in the process of progression (5) (p. 490).

It is also true that the limb extensors are as active in man in progression as in the counteracting of gravity, for if the leg were not carried forward from the maximum of the flexion phase to the forward position normal progression could not take place.

A hemiplegic who cannot stand can swim, and a child who cannot walk can, if suspended in a bath, progress. The suspension band is merely put on to counteract the difference between the specific gravity of the child and that of water, a difference which is practically absent in fish. The idea that standing, counteracting gravity, preceded progression in man is not true. A child fails to walk, not because he cannot progress, but because he cannot stand; not because he cannot move from one place to another, but because walking demands not only the aquatic ability to progress but also the terrestrial ability to counteract gravity. As I have shown in my analysis of gait in man (6), there are four phases, two fin-like and residua of aquatic progression and consisting of the moving of the legs considered as rods forward and backward by corresponding opposite sets of muscles acting at the hips (first and second phase), and two other phases, terrestrial in character, by which one limb is lifted from terra firma by the flexion of the leg by a definite set of muscles (third phase), and the other is fixed to resist terra firma, and so gravity, by a correspondingly opposite set of muscles (fourth phase). Furthermore, not only are the extensors of the knee part of the antigravity group, concerned in straightening the leg after it has passed its maximum of flexion, as has been stated above, but they are also concerned in straightening the leg in the support phase. I have shown (6, 7) that in walking the knee is bent at the maximum phase of support and that it is extended in its maximum ventral or backward position, hence it is extended in passing from the support to

the ventral position as it exerts less and less antigravity activity. Whereas the knee extensors are concerned in counteracting gravity by straightening the leg, they are also concerned in progression by the same action. The angle at the knee, when counteracting gravity while standing and that while walking, is not the same (Holt phenomenon) (7). In standing, the knee extensors only act to counteract gravity, while in walking they do both this and form part of the muscle group active in progression.

As I have tried to show, progression both from the point of view of comparative anatomy, phylogeny, and from the point of view of the development of a man (child), ontogeny, preceded antigravity activity. Progression preceded the counteracting of gravity and is still an important function of the so-called "antigravity" limb muscles. Hence the muscles which counteract gravity have other functions.

As to the application of the term to the elevators of the jaws, it also tells but part of their functions, which include the seizure and mastication of food.

It were better to say, therefore, "the antigravity functions of the axial and appendicular extensors and the elevators of the jaws," firstly, because the muscles do not counteract gravity in all animals; secondly, because they do not always do so even in the same animal; and thirdly, because they are also concerned in a great many other acts equally important, such as progression. When the proper impulses set them in the proper position they counteract gravity, otherwise they do not. To speak of them as a group which counteracts gravity, to the exclusion of other groups and as if they always did that and never did anything else, is obviously incorrect.

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